

=> file biosis caba caplus embase japio lifesci medline scisearch

=> e lubitz werner/au

```
E1          3      LUBITZ W D/AU
E2          1      LUBITZ W J/AU
E3         393 --> LUBITZ WERNER/AU
E4          1      LUBITZ WERNER PROF/AU
E5          1      LUBITZ WILLIAM/AU
E6          2      LUBITZ WILLIAM DAVID/AU
E7          1      LUBITZ WOLFANG/AU
E8         420      LUBITZ WOLFGANG/AU
E9          1      LUBITZKI LOTHAR/AU
E10         1      LUBITZOMERO C/AU
E11         7      LUBITZSCH PETER/AU
E12         1      LUBITZSCH WOLFGANG/AU
```

=> s e1-e4 and bacter? and ghost?

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L1         151 ("LUBITZ W D"/AU OR "LUBITZ W J"/AU OR "LUBITZ WERNER"/AU OR
              "LUBITZ WERNER PROF"/AU) AND BACTER? AND GHOST?
```

=> dup rem l1

PROCESSING COMPLETED FOR L1

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L2          57 DUP REM L1 (94 DUPLICATES REMOVED)
```

=> s bioaffinity and (binding pair)

```
L4          1 BIOAFFINITY AND (BINDING PAIR)
```

=> d

L4 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2009 ACS on STN

AN 1990:512008 CAPLUS <<LOGINID::20091202>>

DN 113:112008

OREF 113:18897a,18900a

TI Application of poly(ethyleneimine) derivatized with a hydrophobic group in
protein immobilization for immunoassays and ***bioaffinity***
separations

IN Lau, Philip Hon Peng

PA du Pont de Nemours, E. I., and Co., USA

SO Eur. Pat. Appl., 12 pp.

CODEN: EPXXDW

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	----	-----	-----	-----
PI	EP 341498	A1	19891115	EP 1989-107607	19890427
	EP 341498	B1	19940518		
	R: DE, FR, GB, IT				
	US 4952519	A	19900828	US 1988-188956	19880502
	JP 02043947	A	19900214	JP 1989-112314	19890502
	JP 07034859	B	19950419		
PRAI	US 1988-188956	A	19880502		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

OSC.G 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

=> s l2 and (biotin or steptavidin or avidin or hapten or saccharide or lectin
or ligand or receptor)

```
L5          3 L2 AND (BIOTIN OR STEPTAVIDIN OR AVIDIN OR HAPTEN OR SACCHARIDE
```

OR LECTIN OR LIGAND OR RECEPTOR)

=> d 1-

YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2005:120755 CAPLUS <<LOGINID::20091202>>
 DN 142:225686
 TI Sealing of ***bacterial*** ***ghosts*** for drug delivery using
 membrane vesicles and affinity ***ligand*** interactions
 IN ***Lubitz, Werner***
 PA Austria
 SO PCT Int. Appl., 37 pp.
 CODEN: PIXXD2
 DT Patent
 LA German
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005011713	A1	20050210	WO 2004-EP8790	20040805
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	DE 10335796	A1	20050303	DE 2003-10335796	20030805
	AU 2004260620	A1	20050210	AU 2004-260620	20040805
	AU 2004260620	B2	20080124		
	CA 2534612	A1	20050210	CA 2004-2534612	20040805
	EP 1656149	A1	20060517	EP 2004-763831	20040805
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
	NZ 545232	A	20081224	NZ 2004-545232	20040805
	US 20060286126	A1	20061221	US 2006-567426	20060516
PRAI	DE 2003-10335796	A	20030805		
	WO 2004-EP8790	W	20040805		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2000:623585 CAPLUS <<LOGINID::20091202>>
 DN 133:227782
 TI ***Bacterial*** ***ghosts*** as carrier and targeting vehicles
 IN Huter, Veronika; ***Lubitz, Werner***
 PA Austria
 SO Ger. Offen., 10 pp.
 CODEN: GWXXBX
 DT Patent
 LA German
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 19909770	A1	20000907	DE 1999-19909770	19990305
	CA 2370714	A1	20000914	CA 2000-2370714	20000303
	WO 2000053163	A1	20000914	WO 2000-EP1906	20000303
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1158966	A1	20011205	EP 2000-912549	20000303
	EP 1158966	B1	20030611		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2002538198	T	20021112	JP 2000-603652	20000303
	AT 242630	T	20030615	AT 2000-912549	20000303
	NZ 514408	A	20040130	NZ 2000-514408	20000303
	AU 778166	B2	20041118	AU 2000-34272	20000303
PRAI	DE 1999-19909770	A	19990305		
	WO 2000-EP1906	W	20000303		
OSC.G	4	THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)			

L5 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN

AN 1992:1761 CAPLUS <<LOGINID::20091202>>

DN 116:1761

OREF 116:363a,366a

TI Membrane-anchoring of heterologous proteins in recombinant hosts for use as antigens

IN ***Lubitz, Werner*** ; Szostak, Michael P.

PA Boehringer Mannheim G.m.b.H., Germany

SO PCT Int. Appl., 46 pp.

CODEN: PIXXD2

DT Patent

LA German

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9113155	A1	19910905	WO 1991-EP308	19910219
	W: AU, FI, HU, JP, SU, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
	DE 4005874	A1	19911107	DE 1990-4005874	19900224
	AU 9172373	A	19910918	AU 1991-72373	19910219
	EP 516655	A1	19921209	EP 1991-903789	19910219
	EP 516655	B1	19940504		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 05503014	T	19930527	JP 1991-503980	19910219
	JP 3238396	B2	20011210		
	AT 105335	T	19940515	AT 1991-903789	19910219
	US 5470573	A	19951128	US 1992-924028	19920930
PRAI	DE 1990-4005874	A	19900224		
	EP 1991-903789	A	19910219		
	WO 1991-EP308	A	19910219		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s bacter? and ghost? and (biotin or steptavidin or avidin or hapten or
saccharide or lectin or ligand or receptor)
L6 107 BACTER? AND GHOST? AND (BIOTIN OR STEPTAVIDIN OR AVIDIN OR HAPTE
 N OR SACCHARIDE OR LECTIN OR LIGAND OR RECEPTOR)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 68 DUP REM L6 (39 DUPLICATES REMOVED)

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 68 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 2009:589834 BIOSIS <<LOGINID::20091202>>
DN PREV200900590937
TI Pharmaceutical Biotechnology.
AU Guzman, CA [Editor]; Feuerstein, GZ [Editor]
SO Guzman, CA [Editor]; Feuerstein, GZ [Editor]. Adv. Exp. Med. Biol., (2009)
 Pharmaceutical Biotechnology.
 Publisher: SPRINGER-VERLAG BERLIN, HEIDELBERGER PLATZ 3, D-14197 BERLIN,
 GERMANY. Series: Advances in Experimental Medicine and Biology.
 CODEN: AEMBAP. ISSN: 0065-2598. ISBN: 978-1-4419-1131-5(H).
DT Book
LA English
ED Entered STN: 21 Oct 2009
 Last Updated on STN: 21 Oct 2009
AB This 254-page book presents and describes Pharmaceutical Biotechnology.
 The book is organized into 15 individually authored chapters and these are
 further divided into different sections. The first chapter deals with
 translational medicine, a paradigm shift in modern drug discovery and
 development, the role of biomarkers. The second chapter deals with
 natural products in drug discovery, present status and perspectives. The
 third chapter deals with protein pharmaceuticals, discovery and
 preclinical development. Remaining chapters include the role of
 nanobiotechnology in drug discovery, conotoxin venom peptide therapeutics,
 shark novel antigen receptors, immune interventions of human diseases
 through toll-like receptors, genome-based vaccine development, virus-like
 particles as a vaccine delivery system and applications of
 bacterial ***ghosts*** in biomedicine. The book highlights a
 list of contributors and their respective institutions. Each chapter
 contains a list of references. The text is written in English. Users of
 this book will include biotechnologists, molecular biologists, and
 pharmacologists.
AB. . . interventions of human diseases through toll-like receptors,
 genome-based vaccine development, virus-like particles as a vaccine
 delivery system and applications of ***bacterial*** ***ghosts***
 in biomedicine. The book highlights a list of contributors and their
 respective institutions. Each chapter contains a list of references.. .
 .
IT . . .
 system disease, viral disease
 Influenza (MeSH)

IT Chemicals & Biochemicals
 cytokine; calcium channel; conotoxin; sodium channel; protein
 pharmaceuticals; immune modulators; toll-like ***receptor*** :
 signaling

L7 ANSWER 2 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2008:316903 CAPLUS <<LOGINID::20091202>>

DN 148:306434

TI Use of glycolipids as adjuvants

IN Ebensen, Thomas; Morr, Michael; Guzman, Carlos A.; Milkereit, Goetz

PA Helmholtz-Zentrum Fuer Infektionsforschung GmbH, Germany

SO PCT Int. Appl., 75 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2008028667	A2	20080313	WO 2007-EP7794	20070906
	WO 2008028667	A3	20080703		
	W:				
	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA,				
	CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI,				
	GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG,				
	KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME,				
	MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL,				
	PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN,				
	TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW:				
	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,				
	IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF,				
	BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW,				
	GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,				
	BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
	EP 1897557	A1	20080312	EP 2006-18723	20060907
	R:				
	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,				
	IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL,				
	BA, HR, MK, YU				
	AU 2007294103	A1	20080313	AU 2007-294103	20070906
	CA 2661280	A1	20080313	CA 2007-2661280	20070906
	EP 2059257	A2	20090520	EP 2007-802189	20070906
	R:				
	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,				
	IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR,				
	AL, BA, HR, MK, RS				
	IN 2009MN00464	A	20090515	IN 2009-MN464	20090305
PRAI	EP 2006-18723	A	20060907		
	WO 2007-EP7794	W	20070906		

OS MARPAT 148:306434

AB The present invention relates to adjuvants of the glycolipid type and
 their uses in pharmaceutical compns., like in vaccines. In particular,
 the present invention provides new uses of compds. useful as adjuvants for
 prophylactic and/or therapeutic vaccination in the treatment of infectious
 diseases, inflammatory diseases, autoimmune diseases, tumors and
 allergies. The compds. are particularly useful not only as systemic, but
 preferably as mucosal adjuvants.

IT Glycoproteins

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(CD40-L (antigen CD40 ***ligand***); vaccines comprising antigen
 and glycolipids as adjuvants induce IgG, IgA, and T-cell responses)

IT Eubacteria
(***ghost*** ***bacteria*** , antigen delivery system; vaccines comprising antigen and glycolipids as adjuvants induce IgG, IgA, and T-cell responses)

L7 ANSWER 3 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2008:1185246 CAPLUS <<LOGINID::20091202>>

DN 149:400314

TI Adjuvant combinations comprising a microbial TLR agonist, a CD40 or 4-1BB agonist, and optionally an antigen and the use thereof for inducing a synergistic enhancement in cellular immunity

IN Delucia, Dave

PA Regents of the University of Colorado, USA

SO U.S. Pat. Appl. Publ., 10pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	----	-----	-----	-----
PI	US 20080241139	A1	20081002	US 2007-931237	20071031
PRAI	US 2006-863695P	P	20061031		

AB Adjuvant combinations comprising at least one microbial TLR agonist such as a whole virus, ***bacterium*** or yeast or portion thereof such a membrane, spheroplast, cytoplasm, or ***ghost*** , a CD40 or 4-1BB agonist and optionally an antigen wherein all 3 moieties may be sep. or comprise the same recombinant microorganism or virus are disclosed. The CD40 or 4-1BB agonists preferably comprise an agonistic anti-CD40 antibody or anti-4-1BB antibody. The use of these immune adjuvants for treatment of various chronic diseases such as cancer, allergy, inflammation, infection, and autoimmune disease is also provided.

AB Adjuvant combinations comprising at least one microbial TLR agonist such as a whole virus, ***bacterium*** or yeast or portion thereof such a membrane, spheroplast, cytoplasm, or ***ghost*** , a CD40 or 4-1BB agonist and optionally an antigen wherein all 3 moieties may be sep. or comprise the same. . .

IT Glycoproteins

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(CD40-L (antigen CD40 ***ligand***); adjuvant combinations comprising a microbial TLR agonist, a CD40 or 4-1BB agonist, and antigen and use thereof for inducing a synergistic enhancement in cellular immunity)

L7 ANSWER 4 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

AN 2008482706 EMBASE <<LOGINID::20091202>>

TI Preventing recurrent urinary tract infections: Role of vaccines.

AU Zakri, Rhana Hassan; DasGupta, Ranan; Dasgupta, Prokar; Khan, Mohammad Shamim

CS Department of Urology, Guy's and St. Thomas' NHS Foundation Trust, King's London School of Medicine, London, United Kingdom. rhzakri@doctors.org.uk

AU Zakri, Rhana Hassan

CS Urology, Guy's and St. Thomas' NHS Foundation Trust, Great Maze Pond, London SE1 9RJ, United Kingdom. rhzakri@doctors.org.uk

AU Zakri, R. H. (correspondence)

CS Urology, Guy's and St. Thomas' NHS Foundation Trust, Great Maze Pond, London SE1 9RJ, United Kingdom. rhzakri@doctors.org.uk

SO BJU International, (November 2008) Vol. 102, No. 9, pp. 1055-1056.
 Refs: 12
 ISSN: 1464-4096; E-ISSN: 1464-410X CODEN: BJINFO

PB Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG, United Kingdom.

CY United Kingdom

DT Journal; Note

FS 004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
 017 Public Health, Social Medicine and Epidemiology
 028 Urology and Nephrology
 037 Drug Literature Index
 038 Adverse Reactions Titles

LA English

ED Entered STN: 4 Nov 2008
 Last Updated on STN: 4 Nov 2008

CT Medical Descriptors:
 bacterial strain
 clinical trial
 Escherichia coli
 follow up
 human
 hysterectomy
 nonhuman
 note
 priority journal
 prophylaxis
 pyelonephritis: DT, drug therapy
 pyelonephritis: PC, prevention
 recurrent disease
 spinal cord injury
 unspecified side effect: SI, side effect
 upregulation
 *urinary tract infection: DT, drug therapy
 *urinary tract infection: PC, prevention
 vaccination
 vagina mucosa
 bacterial protein: EC, endogenous compound
 ****bacterial vaccine: DT, drug therapy***
 ****bacterial vaccine: NA, intranasal drug administration***
 ****bacterial vaccine: PD, pharmacology***
 ****bacterial vaccine: SC, subcutaneous drug administration***
 immunoglobulin G: EC, endogenous compound
 outer membrane protein A: EC, endogenous compound
 *papdg vaccine: DT, drug therapy
 placebo
 protein fepa: EC, endogenous compound
 ****siderophore receptor iron: PD, pharmacology***
 ****siderophore receptor iron: SC, subcutaneous drug administration***
 *solco urovac: AE, adverse drug reaction
 *solco urovac: CT, clinical trial
 *solco urovac: DT, drug therapy
 *solco urovac: PA, parenteral drug administration
 unclassified drug
 ****vibrio cholerae ghosts vaccine: NA, intranasal drug***
 *** administration***

AN 2007:1469963 CAPLUS <<LOGINID::20091202>>
 DN 148:99092
 TI Immunogenic multivalent adhesins preparation and use as vaccines
 IN Knight, Stefan
 PA Swed.
 SO PCT Int. Appl., 40pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2007148229	A2	20071227	WO 2007-IB2430	20070222
	WO 2007148229	A3	20081030		
	W:				
	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW:				
	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				

PRAI US 2006-775678P P 20060222

AB Immunogenic multivalent complexes comprise a ***receptor*** -binding domain of a two-domain adhesin antigen or a single chain polyadhesin antigen coupled with a carrier particle. The antigen and the carrier particle are coupled by an affinity-tag system that uses a small peptide tag and that allows a 1-step affinity purifn. The immunogenic multivalent complex further comprises a flexible linker between the C terminus of the antigen and the coupling tag. Immunostimulating and adhesion-blocking agents, vaccines, immunogenic formulations, and immunogenic constructs and compns. comprise an immunogenic multivalent complex. Methods for identifying a two-domain adhesin antigen comprise selecting a sequence of a pilin from a chaperone/usher system; searching a protein and/or DNA sequence database with the pilin sequence; and identifying a sequence that aligns to the C-terminal portion of the pilin sequence and comprises an unmatched sequence of from about 140 to about 240 amino acid residues preceding the aligned region.

AB Immunogenic multivalent complexes comprise a ***receptor*** -binding domain of a two-domain adhesin antigen or a single chain polyadhesin antigen coupled with a carrier particle. The antigen and. . .

IT Eubacteria
 (***ghosts*** , as carrier particles; immunogenic multivalent adhesins prepn. and use as vaccines)

IT Affinity chromatography
 Bacterial infection
 Gram-negative ***bacteria***
 Linking agents
 Pharmaceutical carriers
 Vaccines
 (immunogenic multivalent adhesins prepn. and use as vaccines)

AN 2007:1171779 CAPLUS <<LOGINID::20091202>>
 DN 147:467781
 TI Her-2/neu multi-peptide cancer vaccine
 IN Zielinski, Christoph; Schreiner, Otto; Pehamberger, Hubert; Breiteneder, Heimo; Wiedermann, Ursula
 PA Bio Life Science Forschungs- und Entwicklungsges.m.b.H., Austria
 SO Eur. Pat. Appl., 26pp.
 CODEN: EPXXDW
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1844788	A1	20071017	EP 2006-7834	20060413
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
	AU 2007237491	A1	20071025	AU 2007-237491	20070411
	CA 2649013	A1	20071025	CA 2007-2649013	20070411
	WO 2007118660	A2	20071025	WO 2007-EP3226	20070411
	WO 2007118660	A3	20071213		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
	EP 2004218	A2	20081224	EP 2007-724167	20070411
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR				
	US 20090269364	A1	20091029	US 2009-296738	20090403
PRAI	EP 2006-7834	A	20060413		
	WO 2007-EP3226	W	20070411		

AB A multi-peptide multiepitope vaccine against cancers assocd. with HER-2/neu oncogene overexpression is disclosed. The vaccine comprises a specific combination of peptides presenting different amino acids sequences that are present in the extracellular domain of HER-2/neu protein. The inventors demonstrate that the above vaccine is effective in preventing neu-expressing tumors and that the effect could be increased by co-administration of interleukin-12. Also, the vaccine could be administered as a mucosal vaccine without losing its high immunogenicity, which would be an attractive vaccine for tumors located at mucosal surfaces.

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT Drug delivery systems
 (***bacterial*** ***ghosts*** , vaccine carriers; her-2/neu multi-peptide cancer vaccine)
 IT Interleukin 2
 Interleukin 4
 neu (***receptor***)

RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (her-2/neu multi-peptide cancer vaccine)
 IT Lactic acid ***bacteria***
 (mucosal adjuvants; her-2/neu multi-peptide cancer vaccine)

L7 ANSWER 7 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2007:502112 CAPLUS <<LOGINID::20091202>>
 DN 146:480526
 TI Psudomonas quinolone signal and c-diGMP and conjugate as mucosal adjuvant
 for vaccine preparation against infection, autoimmune disease,
 inflammation, allergy, cancer and for fertility control
 IN Ebensen, Thomas; Morr, Michael; Guzman, Carlos A.
 PA GBF Gesellschaft fuer Biotechnologische Forschung mbH, Germany
 SO Eur. Pat. Appl., 43pp.
 CODEN: EPXXDW
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1782826	A1	20070509	EP 2005-24266	20051108
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
	AU 2006312688	A1	20070518	AU 2006-312688	20061108
	AU 2006312692	A1	20070518	AU 2006-312692	20061108
	CA 2624903	A1	20070518	CA 2006-2624903	20061108
	CA 2624905	A1	20070518	CA 2006-2624905	20061108
	WO 2007054279	A2	20070518	WO 2006-EP10693	20061108
	WO 2007054279	A3	20070830		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
	WO 2007054283	A2	20070518	WO 2006-EP10699	20061108
	WO 2007054283	A3	20070809		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
	EP 1959989	A2	20080827	EP 2006-806710	20061108

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
 IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR
 EP 1959990 A2 20080827 EP 2006-828961 20061108
 R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
 IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR
 US 20080286296 A1 20081120 US 2008-92747 20080506
 IN 2008MN00958 A 20080718 IN 2008-MN958 20080512
 US 20090169609 A1 20090702 US 2008-92518 20080825
 PRAI EP 2005-24266 A 20051108
 WO 2006-EP10693 W 20061108
 WO 2006-EP10699 W 20061108
 OS MARPAT 146:480526
 AB The present invention relates to new adjuvants and the uses in
 pharmaceutical compns., such as in vaccines. In particular, the present
 invention provides new compds. useful as adjuvants and/or immunomodulators
 for prophylactic and/or therapeutic vaccination in the treatment of
 infectious diseases, inflammatory diseases, autoimmune diseases, tumors,
 and allergies as well as for the control of fertility in human or animal
 populations. The compds. are particularly useful not only as systemic
 agents, but preferably as mucosal adjuvants. In addn., the invention
 relates to its uses as active ingredients in pharmaceutical compns.
 RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
 IT Glycoproteins
 RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)
 (CD40-L (antigen CD40 ***ligand***); Pseudomonas quinolone signal
 and c-diGMP and conjugate as mucosal adjuvant for vaccine prepn.
 against infection, autoimmune disease, inflammation, allergy, and
 cancer and for fertility control)
 IT Eubacteria
 (***ghost*** ***bacteria*** ; Pseudomonas quinolone signal and
 c-diGMP and conjugate as mucosal adjuvant for vaccine prepn. against
 infection, autoimmune disease, inflammation, allergy, and cancer and
 for fertility control)
 L7 ANSWER 8 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights
 reserved on STN
 AN 2007370158 EMBASE <<LOGINID::20091202>>
 TI Recent advances in delivery systems for anti-HIV1 therapy.
 AU Lanao, Jose M. (correspondence); Briones, Elsa; Colino, Clara I.
 CS Department of Pharmacy and Pharmaceutical Technology, Faculty of Pharmacy,
 University of Salamanca, Salamanca, Spain.
 SO Journal of Drug Targeting, (Jan 2007) Vol. 15, No. 1, pp. 21-36.
 Refs: 155
 ISSN: 1061-186X; E-ISSN: 1029-2330 CODEN: JDTAEH
 PUI 772692962
 CY United Kingdom
 DT Journal; Article
 FS 026 Immunology, Serology and Transplantation
 030 Clinical and Experimental Pharmacology
 037 Drug Literature Index
 038 Adverse Reactions Titles
 039 Pharmacy
 004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
 LA English
 SL English

ED Entered STN: 24 Aug 2007
 Last Updated on STN: 24 Aug 2007

AB In the last years, different non-biological and biological carrier systems have been developed for anti-HIV1 therapy. Liposomes are excellent potential anti-HIV1 carriers that have been tested with drugs, antisense oligonucleotides, ribozymes and therapeutic genes. Nanoparticles and low-density lipoproteins (LDLs) are cell-specific transporters of drugs against macrophage-specific infections such as HIV1. Through a process of protein transduction, cell-permeable peptides of natural origin or designed artificially allow the delivery of drugs and genetic material inside the cell. Erythrocyte ***ghosts*** and ***bacterial*** ***ghosts*** are a promising delivery system for therapeutic peptides and HIV vaccines. Of interest are the advances made in the field of HIV gene therapy by the use of autologous haematopoietic stem cells and viral vectors for HIV vaccines. Although important milestones have been reached in the development of carrier systems for the treatment of HIV, especially in the field of gene therapy, further clinical trials are required so that the efficiency and safety of these new systems can be guaranteed in HIV patients.

AB . . . cell-permeable peptides of natural origin or designed artificially allow the delivery of drugs and genetic material inside the cell. Erythrocyte ***ghosts*** and ***bacterial*** ***ghosts*** are a promising delivery system for therapeutic peptides and HIV vaccines. Of interest are the advances made in the field. . .

CT Medical Descriptors:
 Adenovirus
 anemia: SI, side effect
 antiviral activity
 article
 bacterial membrane
 biodegradability
 bone marrow toxicity: SI, side effect
 CD4+ T lymphocyte
 cellular immunity
 drug accumulation
 drug blood level
 drug delivery system
 drug half life
 drug receptor binding
 drug safety
 encapsulation
 erythrocyte ghost
 human
 Human immunodeficiency virus 1
 *Human immunodeficiency virus 1 infection: DT, drug therapy
 humoral immunity
 leukopenia: SI, side effect
 liposomal gene delivery system
 nonhuman
 nonviral gene delivery. . .
 2 methylpiperazine: PD, pharmacology
 aciclovir: PR, pharmaceuticals
 aciclovir: PD, pharmacology
 adefovir: PD, pharmacology
 antisense oligonucleotide: PR, pharmaceuticals
 antisense oligonucleotide: PD, pharmacology
 aplavirok: DV, drug development

aplavirok: PD, pharmacology
 chemokine receptor CCR5 antagonist: DV, drug development
 chemokine receptor CCR5 antagonist: PD, pharmacology
 didanosine: CB, drug combination
 didanosine: PR, pharmaceuticals
 DNA vaccine: PR, pharmaceuticals
 flucytosine: PR, pharmaceuticals
 flucytosine: PD, pharmacology
 ganciclovir: PR, pharmaceuticals
 glutathione: CB, . . .

L7 ANSWER 9 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2006:1124271 CAPLUS <<LOGINID::20091202>>

DN 145:434372

TI Nanosized biological container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manufacture thereof

IN Chen, Liaohai; Bader, Samuel D.; Hoffmann, Axel F.; Kay, Brian K.; Makowski, Lee

PA The University of Chicago, USA

SO U.S. Pat. Appl. Publ., 27 pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	---	-----	-----	-----
PI	US 20060240456	A1	20061026	US 2006-384792	20060320
PRAI	US 2005-664235P	P	20050322		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Nanosized biol. containers that are " ***ghosts*** " of viruses for which capsids are independent of their endogenous viral nucleic acid cores, provide nano-particles of uniform size, and known nos. of sites for attachments of ligands. These containers can be filled with a fluorescent, magnetic, x-ray absorbent, nucleotide components or a radioactive particle and used as nanoscale markers.

AB Nanosized biol. containers that are " ***ghosts*** " of viruses for which capsids are independent of their endogenous viral nucleic acid cores, provide nano-particles of uniform size, and. . .

IT Fusion proteins (chimeric proteins)

RL: BSU (Biological study, unclassified); DEV (Device component use); BIOL (Biological study); USES (Uses)

(capsid protein comprising a ***ligand*** ; nanosized biol. container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manuf. thereof)

IT Antibodies and Immunoglobulins

RL: BSU (Biological study, unclassified); DEV (Device component use); BIOL (Biological study); USES (Uses)

(***ligand*** ; nanosized biol. container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manuf. thereof)

IT ***Bacteriophage***

Buffers

Fluorescent substances

Magnetic particles

Phage display library

Virus

(nanosized biol. container composed of virus capsid proteins, filled with fluorescent, magnetic, x-ray absorbent, nucleotide components and manuf. thereof)

L7 ANSWER 10 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 1

AN 2006:510281 BIOSIS <<LOGINID::20091202>>

DN PREV200600513565

TI A mechanism of cell death involving an adenylyl cyclase/PKA signaling pathway is induced by the Cry1Ab toxin of *Bacillus thuringiensis*.

AU Zhang, Xuebin; Candas, Mehmet; Griko, Natalya B.; Taussig, Ronald; Bulla, Lee A. Jr. [Reprint Author]

CS Univ Texas, Dept Mol and Cell Biol, Richardson, TX 75083 USA
lee.bulla@utdallas.edu

SO Proceedings of the National Academy of Sciences of the United States of America, (JUN 27 2006) Vol. 103, No. 26, pp. 9897-9902.
CODEN: PNASA6. ISSN: 0027-8424.

DT Article

LA English

ED Entered STN: 4 Oct 2006
Last Updated on STN: 4 Oct 2006

AB Many pathogenic organisms and their toxins target host cell receptors, the consequence of which is altered signaling events that lead to aberrant activity or cell death. A significant body of literature describes various molecular and cellular aspects of toxins associated with ***bacterial*** invasion, colonization, and host cell disruption. However, there is little information on the molecular and cellular mechanisms associated with the insecticidal action of *Bacillus thuringiensis* (Bt) Cry toxins. Recently, we reported that the Cry1Ab toxin produced by Bt kills insect cells by activating a Mg²⁺-dependent cytotoxic event upon binding of the toxin to its ***receptor*** BT-R-1. Here we show that binding of Cry toxin to BT-R1 provokes cell death by activating a previously undescribed signaling pathway involving stimulation of G protein (G(alpha s)) and adenylyl cyclase, increased cAMP levels, and activation of protein kinase A. Induction of the adenylyl cyclase/protein kinase A pathway is manifested by sequential cytological changes that include membrane blebbing, appearance of ***ghost*** nuclei, cell swelling, and lysis. The discovery of a toxin-induced cell death pathway specifically linked to BT-R-1 in insect cells should provide insights into how insects evolve resistance to Bt and into the development of new, safer insecticides.

AB. . . aberrant activity or cell death. A significant body of literature describes various molecular and cellular aspects of toxins associated with ***bacterial*** invasion, colonization, and host cell disruption. However, there is little information on the molecular and cellular mechanisms associated with the. . . toxin produced by Bt kills insect cells by activating a Mg²⁺-dependent cytotoxic event upon binding of the toxin to its ***receptor*** BT-R-1. Here we show that binding of Cry toxin to BT-R1 provokes cell death by activating a previously undescribed signaling. . . Induction of the adenylyl cyclase/protein kinase A pathway is manifested by sequential cytological changes that include membrane blebbing, appearance of ***ghost*** nuclei, cell swelling, and lysis. The discovery of a toxin-induced cell death pathway specifically linked to BT-R-1 in insect cells. . .

IT . . .

Biochemicals
magnesium ion; cyclic AMP; Cry1Ab toxin: toxin; PKA [protein kinase A]:

signaling; adenylyl cyclase [EC 4.6.1.1]: signaling; BT-R-1 [Bt
 receptor]

ORGN Classifier
 Endospore-forming Gram-Positives 07810
 Super Taxa
 Eubacteria; ***Bacteria*** ; Microorganisms
 Organism Name
 Bacillus thuringiensis (species)
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Insecta 75300
 Super Taxa
 Arthropoda; Invertebrata; Animalia
 Organism Name
 H5 cell line (cell_line): insect cells
 Taxa. . .

L7 ANSWER 11 OF 68 CABA COPYRIGHT 2009 CABI on STN
 AN 2008:83068 CABA <<LOGINID::20091202>>
 DN 20063203692
 TI Advances in vaccine development against enterohemorrhagic Escherichia coli
 O157:H7
 AU Liu YanQing; Mao XuHu; Zou QuanMing; Liu, Y. Q.; Mao, X. H.; Zou, Q. M.
 CS Clinical Microbiology and Immunology, The Third Medical University of PLA,
 Chongqing 400038, China. mxh95xy@mail.tmmu.com.cn
 SO Chinese Journal of Zoonoses, (2006) Vol. 22, No. 10, pp. 998-1000. 23 ref.
 Publisher: Editorial Committee of Chinese Journal of Zoonoses, Health and
 Anti-epidemic Station of Fujian Province. Fuzhou
 ISSN: 1002-2694
 URL: <http://www.zgrsghbzz.periodicals.net.cn>

CY China
 DT Journal
 LA Chinese
 ED Entered STN: 5 May 2008
 Last Updated on STN: 5 May 2008

AB Vaccine related protective antigens of enterohemorrhagic Escherichia coli
 O157:H7 include adhesion antigens (e.g. intimin, translocated intimin
 receptor and type III secretion system related protein EspA) and
 toxic antigens. Polysaccharide vaccine, subunit vaccine, transgenic plant
 vaccine and ***bacterial*** ***ghost*** vaccine have been
 developed. Some vaccines have already been put into clinical trials.

AB Vaccine related protective antigens of enterohemorrhagic Escherichia coli
 O157:H7 include adhesion antigens (e.g. intimin, translocated intimin
 receptor and type III secretion system related protein EspA) and
 toxic antigens. Polysaccharide vaccine, subunit vaccine, transgenic plant
 vaccine and ***bacterial*** ***ghost*** vaccine have been
 developed. Some vaccines have already been put into clinical trials.

BT Escherichia; Enterobacteriaceae; Gracilicutes; ***bacteria*** ;
 prokaryotes

L7 ANSWER 12 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2005:120755 CAPLUS <<LOGINID::20091202>>
 DN 142:225686
 TI Sealing of ***bacterial*** ***ghosts*** for drug delivery using
 membrane vesicles and affinity ***ligand*** interactions
 IN Lubitz, Werner

PA Austria
 SO PCT Int. Appl., 37 pp.
 CODEN: PIXXD2
 DT Patent
 LA German
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005011713	A1	20050210	WO 2004-EP8790	20040805
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	DE 10335796	A1	20050303	DE 2003-10335796	20030805
	AU 2004260620	A1	20050210	AU 2004-260620	20040805
	AU 2004260620	B2	20080124		
	CA 2534612	A1	20050210	CA 2004-2534612	20040805
	EP 1656149	A1	20060517	EP 2004-763831	20040805
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
	NZ 545232	A	20081224	NZ 2004-545232	20040805
	US 20060286126	A1	20061221	US 2006-567426	20060516
PRAI	DE 2003-10335796	A	20030805		
	WO 2004-EP8790	W	20040805		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention relates to a method for producing sealed ***bacterial***
 ghosts using the specific interaction between partners of a binding pair. The ***ghosts*** can be loaded with therapeutically useful substances and used as carriers. The inventive sealed ***ghosts*** can be used in medicine, agriculture, and biotechnol. ***Ghosts*** are formed by inducing expression of the E gene, which causes membrane lysis. The ***ghosts*** are then derivatized with a member of a binding pair, e.g. ***biotin***, or a streptavidin-binding peptide. Biotinylation may be via an enzymic biotinylation site incorporated into the E gene product. The derivatized ***ghosts*** are then mixed with lipid vesicles present the other member of the binding pair, e.g. streptavidin. The interaction results in the binding of the lipid vesicles to the ***ghosts***. Sealed ***ghosts*** can be captured using the ***ligand*** immobilized on a suitable carrier.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions

AB The invention relates to a method for producing sealed ***bacterial***
 ghosts using the specific interaction between partners of a binding pair. The ***ghosts*** can be loaded with therapeutically useful substances and used as carriers. The inventive sealed ***ghosts*** can be used in medicine, agriculture, and biotechnol. ***Ghosts*** are formed by inducing expression of the E gene, which causes membrane lysis. The ***ghosts*** are then derivatized with a

member of a binding pair, e.g. ***biotin*** , or a streptavidin-binding peptide. Biotinylation may be via an enzymic biotinylation site incorporated into the E gene product. The derivatized ***ghosts*** are then mixed with lipid vesicles present the other member of the binding pair, e.g. streptavidin. The interaction results in the binding of the lipid vesicles to the ***ghosts*** . Sealed ***ghosts*** can be captured using the ***ligand*** immobilized on a suitable carrier.

ST ***bacteria*** membrane ***ghost*** sealing lipid vesicle affinity interaction; membrane ***biotin*** vesicle streptavidin
bacteria ***ghost*** sealing

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(E; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Drug delivery systems
(***bacterial*** ***ghosts*** as; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Transformation, genetic
(***bacterial*** ***ghosts*** for delivery of nucleic acids in; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Agrochemicals
Drugs
Dyes
Organelle
(***bacterial*** ***ghosts*** for delivery of; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Nucleic acids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(***bacterial*** ***ghosts*** for delivery of; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Protein motifs
(biotinylation, lysis proteins contg.; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Protoplast and Spheroplast
(cell ***ghost*** ; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Virion structure
(envelope, sealing of membrane ***ghosts*** with; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Antibodies and Immunoglobulins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(fragments, in affinity binding of membrane vesicles to ***bacterial*** ***ghosts*** ; sealing of ***bacterial*** ***ghosts*** for drug delivery using membrane vesicles and affinity ***ligand*** interactions)

IT Agglutinins and Lectins
Antibodies and Immunoglobulins
Avidins

Carbohydrates, biological studies
Haptens
Receptors
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
(in affinity binding of membrane vesicles to ***bacterial***
ghosts ; sealing of ***bacterial*** ***ghosts*** for
drug delivery using membrane vesicles and affinity ***ligand***
interactions)

IT Eubacteria
(membrane ***ghosts*** ; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT Proteins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
(membrane, incorporation into ***bacterial*** ***ghosts*** of;
sealing of ***bacterial*** ***ghosts*** for drug delivery using
membrane vesicles and affinity ***ligand*** interactions)

IT Immobilization, molecular or cellular
(of ***bacterial*** ***ghosts*** ; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT Gram-negative ***bacteria***
(prepn. of membrane ***ghosts*** from; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT Agriculture and Agricultural chemistry
Biotechnology
Medicine
(sealing of ***bacterial*** ***ghosts*** for drug delivery
using membrane vesicles and affinity ***ligand*** interactions)

IT Liposomes
(sealing of membrane ***ghosts*** with; sealing of
bacterial ***ghosts*** for drug delivery using membrane
vesicles and affinity ***ligand*** interactions)

IT Lipids, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
(vesicles, sealing of membrane ***ghosts*** with; sealing of
bacterial ***ghosts*** for drug delivery using membrane
vesicles and affinity ***ligand*** interactions)

IT 58-85-5D, ***Biotin*** , analogs, conjugates with proteins 9013-20-1,
Streptavidin
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
(in affinity binding of membrane vesicles to ***bacterial***
ghosts ; sealing of ***bacterial*** ***ghosts*** for
drug delivery using membrane vesicles and affinity ***ligand***
interactions)

IT 842177-75-7 842177-76-8 842177-77-9 842177-78-0 842177-79-1
842177-80-4
RL: PRP (Properties)
(unclaimed nucleotide sequence; sealing of ***bacterial***
ghosts for drug delivery using membrane vesicles and affinity
ligand interactions)

IT 842138-49-2

RL: PRP (Properties)
(unclaimed sequence; sealing of ***bacterial*** ***ghosts***
for drug delivery using membrane vesicles and affinity ***ligand***
interactions)

L7 ANSWER 13 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN DUPLICATE 2
AN 2005434767 EMBASE <<LOGINID::20091202>>
TI A novel fluorescent probe: Europium complex hybridized T7 phage.
AU Liu, Chin-Mei; Jin, Qiaoling; Sutton, April; Chen, Liaohai
(correspondence)
CS Biosciences Division, Argonne National Laboratory, 9700 South Cass Avenue, Argonne, IL 60439, United States. lhchen@anl.gov
SO Bioconjugate Chemistry, (Sep 2005) Vol. 16, No. 5, pp. 1054-1057.
Refs: 16
ISSN: 1043-1802 CODEN: BCCHEs
CY United States
DT Journal; Article
FS 023 Nuclear Medicine
037 Drug Literature Index
039 Pharmacy
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
LA English
SL English
ED Entered STN: 27 Oct 2005
Last Updated on STN: 27 Oct 2005
AB We report on the creation of a novel fluorescent probe of europium-complex hybridized T7 phage. It was made by filling a ***ligand*** -displayed T7 ***ghost*** phage with a fluorescent europium complex particle. The structure of the hybridized phage, which contains a fluorescent inorganic core surrounded by a ***ligand*** -displayed capsid shell, was confirmed by electron microscope, energy-dispersive X-ray analysis (EDX), bioassays, and fluorescence spectrometer. More importantly, as a benefit of the phage display technology, the hybridized phage has the capability to integrate an affinity reagent against virtually any target molecules. The approach provides an original method to fluorescently "tag" a bioligand and/or to "biofunctionalize" a fluorophore particle. By using other types of materials such as radioactive or magnetic particles to fill the ***ghost*** phage, we envision that the hybridized phages represent a new class of fluorescent, magnetic, or radioprobes for imaging and bioassays and could be used both in vitro and in vivo. .COPYRG. 2005 American Chemical Society.
AB . . . report on the creation of a novel fluorescent probe of europium-complex hybridized T7 phage. It was made by filling a ***ligand*** -displayed T7 ***ghost*** phage with a fluorescent europium complex particle. The structure of the hybridized phage, which contains a fluorescent inorganic core surrounded by a ***ligand*** -displayed capsid shell, was confirmed by electron microscope, energy-dispersive X-ray analysis (EDX), bioassays, and fluorescence spectrometer. More importantly, as a benefit. . . to "biofunctionalize" a fluorophore particle. By using other types of materials such as radioactive or magnetic particles to fill the ***ghost*** phage, we envision that the hybridized phages represent a new class of fluorescent, magnetic, or radioprobes for imaging and bioassays. . .
CT Medical Descriptors:
article

bacteriophage T7

bioassay
complex formation
electron microscopy
fluorescence
hybridization
imaging
spectrofluorometry
X ray analysis
*europium: DV, drug development
*europium: PR, pharmaceuticals
*fluorescent dye: DV, drug development
*fluorescent dye: PR, pharmaceuticals
lanthanide: DV, drug development
lanthanide: PR, pharmaceuticals
ligand: DV, drug development
ligand: PR, pharmaceuticals
radioactive material: DV, drug development
radioactive material: PR, pharmaceuticals

L7 ANSWER 14 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 3

AN 2005:112300 BIOSIS <<LOGINID::20091202>>

DN PREV200500114316

TI Translocation of histone proteins across lipid bilayers and mycoplasma
membranes.

AU Rosenbluh, Joseph; Hariton-Gazal, Elana; Dagan, Arie; Rottem, Shlomo;
Graessmann, Adolf; Loyter, Abraham [Reprint Author]

CS Alexander Silberman Inst Life SciDept Biol Chem, Hebrew Univ Jerusalem,
IL-91904, Jerusalem, Israel
loyter@mail.ls.huji.ac.il

SO Journal of Molecular Biology, (January 14 2005) Vol. 345, No. 2, pp.
387-400. print.
ISSN: 0022-2836 (ISSN print).

DT Article

LA English

ED Entered STN: 23 Mar 2005

Last Updated on STN: 23 Mar 2005

AB We show that the three core histones H2A, H3 and H4 can transverse lipid
bilayers of large unilamellar vesicles (LUVs) and multilamellar vesicles
(MLVs). In contrast, the histone H2B, although able to bind to the
liposomes, fails to penetrate the unilamellar and the multilamellar
vesicles. Translocation across the lipid bilayer was determined using
biotin -labeled histones and an ELISA-based system. Following
incubation with the liposomes, external membrane-bound ***biotin***
molecules were neutralized by the addition of ***avidin*** .
Penetrating ***biotin*** -histone conjugates were exposed by Triton
treatment of the neutralized liposomes. The intra-liposomal
biotin -histone conjugates, in contrast to those attached only to
the external surface, were attached to the detergent lysed lipid
molecules. Thus, biotinylated histone molecules that were exposed only
following detergent treatment of the liposomes were considered to be
located at the inner leaflet of the lipid bilayers. The penetrating
histone molecules failed to mediate translocation of BSA molecules
covalently attached to them. Translocation of the core histones,
including H2B, was also observed across mycoplasma cell membranes. The
extent of this translocation was inversely related to the degree of

membrane cholesterol. The addition of cholesterol also reduced the extent of histone penetration into the MLVs. Although able to bind biotinylated histones, human erythrocytes, erythrocyte ***ghosts*** and Escherichia coli cells were impermeable to them. Based on the present and previous data histones appear to be characterized by the same features that characterize cell penetrating peptides and proteins (CPPs). Copyright 2004 Elsevier Ltd. All rights reserved.

AB. . . to the liposomes, fails to penetrate the unilamellar and the multilamellar vesicles. Translocation across the lipid bilayer was determined using ***biotin*** -labeled histones and an ELISA-based system. Following incubation with the liposomes, external membrane-bound ***biotin*** molecules were neutralized by the addition of ***avidin***. Penetrating ***biotin*** -histone conjugates were exposed by Triton treatment of the neutralized liposomes. The intra-liposomal ***biotin*** -histone conjugates, in contrast to those attached only to the external surface, were attached to the detergent lysed lipid molecules. Thus, . . . cholesterol also reduced the extent of histone penetration into the MLVs. Although able to bind biotinylated histones, human erythrocytes, erythrocyte ***ghosts*** and Escherichia coli cells were impermeable to them. Based on the present and previous data histones appear to be characterized. . .

IT . . .

IT Parts, Structures, & Systems of Organisms
erythrocyte: blood and lymphatics

IT Chemicals & Biochemicals
H2A; H3 histone; H4 histone; ***avidin*** ; cell-penetrating peptides; histone protein; lipid bilayer; liposome; membrane-bound protein

ORGN Classifier
Enterobacteriaceae 06702
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Organism Name
Escherichia coli (species)
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 15 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 2005:77370 SCISEARCH <<LOGINID::20091202>>

GA The Genuine Article (R) Number: 883WX

TI Rational design of vaccination strategies to promote antigen entry into the MHC class I-restricted presentation pathway

AU Guzman C A (Reprint)

CS GBF German Res Ctr Biotechnol, Div Microbiol, Vaccine Res Grp, Mascheroder Weg 1, D-38124 Braunschweig, Germany (Reprint)

AU Becker P D

CS GBF German Res Ctr Biotechnol, Div Microbiol, Vaccine Res Grp, D-38124 Braunschweig, Germany
E-mail: cag@gbf.de

CYA Germany

SO TRANSFUSION MEDICINE AND HEMOTHERAPY, (2004) Vol. 31, No. 6, pp. 398-411. ISSN: 1660-3796.

PB KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.

DT General Review; Journal

LA English

REC Reference Count: 180
ED Entered STN: 27 Jan 2005
Last Updated on STN: 27 Jan 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cytotoxic CD8+ T lymphocytes (CTLs) constitute one of the main effector mechanisms against tumors and viral infections. CTLs specifically recognize short peptides (8 - 10 residues long) displayed on the surface of 'target' cells, which result from the processing of foreign or abnormal proteins (e. g. virus and tumor proteins) and are bound to major histocompatibility complex (MHC) class I molecules. Virtually all nucleated cells display on their surface fragments of intracellularly produced polypeptides. When there are signs of invasion or transformation, CTLs take control of the situation by destroying these 'labeled' target cells. This is an extremely efficient mechanism. However, the efficient differentiation of naive CD8+ T cells into CTLs is a limiting prerequisite. To achieve this differentiation, dendritic cells (DCs) are critical since only these professional antigen-presenting cells (APCs) can provide not only the peptide presented onto the MHC class I molecules but also the costimulatory signals required for this activation. To this end, DCs take up antigens and degrade them into peptides which are loaded on MHC class I and presented onto the surface to prime specific T lymphocytes. In this review, we summarize the current knowledge on the mechanisms used by professional APCs in the processing and presentation of endogenous and exogenous antigens in the context of MHC class I molecules (i.e. priming and cross-priming). We will also discuss new vaccination strategies that take advantage of these physiological mechanisms in order to improve the elicitation of cytotoxic responses to eliminate intracellular pathogens and tumors.

STP KeyWords Plus (R): COMPLEX CLASS-I; RECOMBINANT LISTERIA-MONOCYTOGENES; CYTOTOXIC T-LYMPHOCYTES; DENDRITIC CELL MATURATION; ***RECEPTOR*** -MEDIATED ENDOCYTOSIS; PROTEIN-CHAPERONED PEPTIDES; EPITOPE PRECURSOR PEPTIDES; EXOGENOUS SOLUBLE-ANTIGEN; ***BACTERIAL*** ***GHOST*** SYSTEM; TOLL-LIKE ***RECEPTOR*** -9

L7 ANSWER 16 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

AN 2003192797 EMBASE <<LOGINID::20091202>>

TI ***Bacterial*** ***ghosts*** as carrier and targeting systems for mucosal antigen delivery.

AU Jalava, Katri (correspondence); Lubitz, Werner

CS BIRD-C GmbH and CoKEG, Schoenborngasse 12, A-1080 Wien, Austria.
jalava@bird-c.com

AU Eko, Francis O.

CS Department of Microbiology, Morehouse School of Medicine, Atlanta, GA, United States.

AU Riedmann, Eva; Lubitz, Werner

CS Inst. of Microbiology and Genetics, University of Vienna, Vienna, Austria.

SO Expert Review of Vaccines, (Feb 2003) Vol. 2, No. 1, pp. 45-51.
Refs: 42
ISSN: 1476-0584 CODEN: ERVXAX

CY United Kingdom

DT Journal; General Review; (Review)

FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology

LA English

SL English

ED Entered STN: 29 May 2003
Last Updated on STN: 29 May 2003

AB The application of new strategies to develop effective vaccines is essential in modern medicine. The ***bacterial*** ***ghost*** system is a novel vaccine delivery system endowed with intrinsic adjuvant properties. ***Bacterial*** ***ghosts*** are nonliving Gram-negative ***bacterial*** cell envelopes devoid of cytoplasmic contents while maintaining their cellular morphology and native surface antigenic structures including bioadhesive properties. They are produced by PhiX174 protein E-mediated lysis of Gram-negative ***bacteria***. The intrinsic adjuvant properties of ***bacterial*** ***ghost*** preparations enhance immune responses against envelope-bound antigens, including T-cell activation and mucosal immunity. Since native and foreign antigens can be expressed in the envelope complex of ***ghosts*** before E- mediated lysis, multiple antigens of various origin can be presented to the immune system simultaneously. In addition, the extended ***bacterial*** ***ghost*** system represents a platform technology for specific targeting of DNA-encoded antigens to primary antigen-presenting cells. The potency, safety and relatively low production cost of ***bacterial*** ***ghosts*** offer a significant technical advantage, especially when used as combination vaccines.

TI ***Bacterial*** ***ghosts*** as carrier and targeting systems for mucosal antigen delivery.

AB The application of new strategies to develop effective vaccines is essential in modern medicine. The ***bacterial*** ***ghost*** system is a novel vaccine delivery system endowed with intrinsic adjuvant properties. ***Bacterial*** ***ghosts*** are nonliving Gram-negative ***bacterial*** cell envelopes devoid of cytoplasmic contents while maintaining their cellular morphology and native surface antigenic structures including bioadhesive properties. They are produced by PhiX174 protein E-mediated lysis of Gram-negative ***bacteria***. The intrinsic adjuvant properties of ***bacterial*** ***ghost*** preparations enhance immune responses against envelope-bound antigens, including T-cell activation and mucosal immunity. Since native and foreign antigens can be expressed in the envelope complex of ***ghosts*** before E- mediated lysis, multiple antigens of various origin can be presented to the immune system simultaneously. In addition, the extended ***bacterial*** ***ghost*** system represents a platform technology for specific targeting of DNA-encoded antigens to primary antigen-presenting cells. The potency, safety and relatively low production cost of ***bacterial*** ***ghosts*** offer a significant technical advantage, especially when used as combination vaccines.

CT Medical Descriptors:
aerosol
antigen expression
antigen presentation
antigen presenting cell
****bacterial infection: DT, drug therapy***
****bacterial infection: PC, prevention***
bacterial membrane
bacterial strain
cell structure
chlamydia: DT, drug therapy
chlamydia: PC, prevention

cholera: DT, drug therapy
 cholera: PC, prevention
 cytoplasm
 *drug delivery system
 fertility
 gene expression
 Gram negative bacterium
 human
 immune response
 immune system
 immunization
 lysis
 mucosal immunity
 nonhuman
 priority journal
 review
 T lymphocyte activation
 aluminum potassium sulfate: PR, pharmaceuticals
 antigen: PR, pharmaceuticals
 bacterial antigen: PR, pharmaceuticals
 ****bacterial vaccine: DT, drug therapy***
 ****bacterial vaccine: PR, pharmaceuticals***
 chlamydia vaccine: DT, drug therapy
 chlamydia vaccine: PR, pharmaceuticals
 cholera vaccine: DT, drug therapy
 cholera vaccine: PO, oral drug administration
 cholera vaccine:. . . DT, drug therapy
 contraceptive vaccine: NA, intranasal drug administration
 contraceptive vaccine: PO, oral drug administration
 contraceptive vaccine: PR, pharmaceuticals
 DNA: PR, pharmaceuticals
 Freund adjuvant: PR, pharmaceuticals
 lectin: PR, pharmaceuticals
 lipid: PR, pharmaceuticals
 liposome: PR, pharmaceuticals
 muramyl dipeptide: PR, pharmaceuticals
 naked DNA: PR, pharmaceuticals
 polyethyleneimine: PR, pharmaceuticals
 polylysine: PR, pharmaceuticals
 polymer: PR, pharmaceuticals
 protein
 protein e
 toxin: PR,. . .

L7 ANSWER 17 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
 STN
 AN 2002:597026 BIOSIS <<LOGINID::20091202>>
 DN PREV200200597026
 TI Immunologic basis for the protective efficacy of Chlamydia vaccines.
 AU Igietseme, J. U. [Reprint author]; Eko, F. O. [Reprint author]; Ananaba,
 G. A.; Moore, T. [Reprint author]; McMillan, L. [Reprint author]; Ramey,
 K. [Reprint author]; Jones, M. [Reprint author]; Zuzewicz, M. A.; He, Q.
 [Reprint author]; Murdin, A.; Black, C.; Lyn, D. A. [Reprint author]
 CS Morehouse School of Medicine, Atlanta, GA, USA
 SO Abstracts of the General Meeting of the American Society for Microbiology,
 (2002) Vol. 102, pp. 195. print.
 Meeting Info.: 102nd General Meeting of the American Society for

Microbiology. Salt Lake City, UT, USA. May 19-23, 2002. American Society for Microbiology.
ISSN: 1060-2011.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 20 Nov 2002
Last Updated on STN: 20 Nov 2002

AB An efficacious vaccine that induces a sterilizing and long-term protective immunity is needed to control the ubiquitous oculogenital diseases caused by the obligate intracellular ***bacterium***, Chlamydia trachomatis. To define the cellular and molecular immunologic basis for the potency of a potentially efficacious vaccine against C. trachomatis, we analyzed a surrogate mouse model system of the genital infection. In this model system, an IL-10KO dendritic cell (DC)-based cellular vaccine confers a sterilizing, long-term protective anti-chlamydial genital immunity while two subunit vaccines (a chlamydial MOMP-ISCOMS preparation and Vibrio cholerae ***ghosts*** expressing chlamydial MOMP) induce a partial, short-term protection. The ability to confer protection correlates with the induction of genital mucosal Th1 response. Analysis of the kinetics of induction and maintenance of mucosal Th1 cells revealed that the DC-based regimen induced a greater (approx5-fold) Th1 response than the MOMP-ISCOMS vaccine. Even at 200 days post immunization, the frequency of specific Th1 cells in the recipients of MOMP-ISCOMS were essentially reduced to the baseline naive mouse level; however, recipients of the DC-based cellular vaccine retained a relatively high Th1 response. The long-term protection from genital infection induced by the DC-based cellular vaccine was associated with the preservation of high frequency of Th1 cells, marked by the presence in the genital mucosa of mononuclear cells bearing the alpha1/beta2, alpha4/beta1, and alpha4/beta7 integrins, and specific antibodies, especially IgG2a. Finally, the dominant role of the Th1 cytokine, IFN-gamma, in protective anti-chlamydial immunity was revealed by the finding that the highly efficacious immune T cells from IL-10KO DC-based cellular vaccine immunized animals were ineffective in protecting IFN-gamma ***receptor*** knockout mice from the acute disease of genital chlamydial infection.

AB. . . induces a sterilizing and long-term protective immunity is needed to control the ubiquitous oculogenital diseases caused by the obligate intracellular ***bacterium***, Chlamydia trachomatis. To define the cellular and molecular immunologic basis for the potency of a potentially efficacious vaccine against C. . . vaccine confers a sterilizing, long-term protective anti-chlamydial genital immunity while two subunit vaccines (a chlamydial MOMP-ISCOMS preparation and Vibrio cholerae ***ghosts*** expressing chlamydial MOMP) induce a partial, short-term protection. The ability to confer protection correlates with the induction of genital mucosal. . . finding that the highly efficacious immune T cells from IL-10KO DC-based cellular vaccine immunized animals were ineffective in protecting IFN-gamma ***receptor*** knockout mice from the acute disease of genital chlamydial infection.

IT Major Concepts
Immune System (Chemical Coordination and Homeostasis); Infection;
Pharmacology

IT Chemicals & Biochemicals
bacterial vaccines: applications, development

ORGN Classifier
Chlamydiaceae 07121
Super Taxa

Chlamydiales; Rickettsias and Chlamydias; Eubacteria; ***Bacteria***
; Microorganisms

Organism Name

Chlamydia spp.: pathogen

Chlamydia trachomatis: pathogen

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

mouse: animal model, host

Taxa. . .

L7 ANSWER 18 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2001:338673 CAPLUS <<LOGINID::20091202>>

DN 134:350284

TI Methods to screen microorganisms or gene libraries for products secreted
from a cell

IN Moeller, Soeren; Kongsbak, Lars; Kristensen, Hans-Henrik; Vind, Jesper;
Pedersen, Henrik; Husum, Tommy Lykke

PA Novozymes A/S, Den.

SO PCT Int. Appl., 55 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	----	-----	-----	-----
PI	WO 2001032829	A2	20010510	WO 2000-DK566	20001010
	WO 2001032829	A3	20011213		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
	AU 2000076460	A	20010514	AU 2000-76460	20001010
PRAI	DK 1999-1602	A	19991105		
	WO 2000-DK566	W	20001010		

AB The invention describes methods for screening for products secreted from the cells, and provides methods to establish a correlation between the activity of the secreted product and the secreting cell. Accordingly in a first aspect the present invention relates to a method for screening a DNA library for DNA of interest comprising the steps of (a) creating host cells comprising the DNA library, (b) generating samples each comprising a host cell of step (a), (c) establishing a means for correlating of interest in a sample of the sample, (d) detg. which intensity interval of fluorescence indicates secretion in the sample when the correlating means of step (c) is used, (e) cultivating the samples under suitable conditions, and (f) selecting the samples exhibiting fluorescence within the intensity interval of step (d) using a fluorescence analyzer; wherein the host cell comprises DNA of interest.

OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT Erythrocyte
Macrophage
(***ghost*** ; methods to screen microorganisms or gene libraries
for products secreted from a cell)

IT Animal tissue culture
Antimicrobial agents
Aspergillus
Aspergillus nidulans
Aspergillus niger
Aspergillus oryzae
Bacillus (***bacterium*** genus)
Bacillus clausii
Bacillus licheniformis
Bacillus subtilis
Bacteria (Eubacteria)
Carbon sources, microbial
Cell
Culture media
DNA sequences
Diffusion
Drugs
Encapsulation
Escherichia
Escherichia coli
Evolution
Films
Fluorescence
Fluorescent substances
Fluorometers
Fungi
Genomic library
Liposomes
Microorganism
Microspheres
Nucleic acid library
Samples
Secretion (process)
(methods to screen microorganisms or gene libraries for products
secreted from a cell)

IT 58-85-5, ***Biotin*** 81-88-9 2321-07-5, Fluorescein 9000-07-1,
Carrageenan 9000-69-5, Pectin 9000-92-4, Amylase 9003-05-8,
Polyacrylamide 9004-34-6, Cellulose, biological studies 9004-54-0,
Dextran, biological studies 9005-25-8, Starch, biological studies
9005-32-7, Alginic acid 9012-36-6, Agarose 9012-76-4, Chitosan
9013-20-1D, Streptavidin., Fluorescently labeled 51306-35-5
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
(methods to screen microorganisms or gene libraries for products
secreted from a cell)

L7 ANSWER 19 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 4

AN 2001:301238 BIOSIS <<LOGINID::20091202>>

DN PREV200100301238

TI Glycophorin as a ***receptor*** for Escherichia coli alpha-hemolysin
 in erythrocytes.
 AU Cortajarena, Aitziber L.; Goni, Felix M.; Ostolaza, Helena [Reprint
 author]
 CS Unidad de Biofisica, Departamento de Bioquimica, Consejo Superior de
 Investigaciones Cientificas-UPV/EHU, Universidad del Pais Vasco/Euskal
 Herriko Unibertsitatea, Bilbao, 48080, Spain
 gbzoseth@lg.ehu.es
 SO Journal of Biological Chemistry, (April 20, 2001) Vol. 276, No. 16, pp.
 12513-12519. print.
 CODEN: JBCHA3. ISSN: 0021-9258.
 DT Article
 LA English
 ED Entered STN: 27 Jun 2001
 Last Updated on STN: 19 Feb 2002
 AB Escherichia coli alpha-hemolysin (HlyA) can lyse both red blood cells
 (RBC) and liposomes. However, the cells are lysed at HlyA concentrations
 1-2 orders of magnitude lower than liposomes (large unilamellar vesicles).
 Treatment of RBC with trypsin, but not with chymotrypsin, reduces the
 sensitivity of RBC toward HlyA to the level of the liposomes. Since
 glycophorin, one of the main proteins in the RBC surface, can be
 hydrolyzed by trypsin much more readily than by chymotrypsin, the
 possibility was tested of a specific binding of HlyA to glycophorin. With
 this purpose, a number of experiments were performed. (a) HlyA was
 preincubated with purified glycophorin, after which it was found to be
 inactive against both RBC and liposomes. (b) Treatment of RBC with an
 anti-glycophorin antibody protected the cells against HlyA lysis. (c)
 Immobilized HlyA was able to bind glycophorin present in a detergent
 lysate of RBC ***ghosts***. (d) Incorporation of glycophorin into pure
 phosphatidylcholine liposomes increased notoriously the sensitivity of the
 vesicles toward HlyA. (e) Treatment of the glycophorin-containing
 liposomes with trypsin reverted the vesicles to their original low
 sensitivity. The above results are interpreted in terms of glycophorin
 acting as a ***receptor*** for HlyA in RBC. The binding constant of
 HlyA for glycophorin was estimated, in RBC at sublytic HlyA
 concentrations, to be 1.5×10^{-9} M.
 TI Glycophorin as a ***receptor*** for Escherichia coli alpha-hemolysin
 in erythrocytes.
 AB. . . the cells against HlyA lysis. (c) Immobilized HlyA was able to bind
 glycophorin present in a detergent lysate of RBC ***ghosts***. (d)
 Incorporation of glycophorin into pure phosphatidylcholine liposomes
 increased notoriously the sensitivity of the vesicles toward HlyA. (e)
 Treatment of. . . reverted the vesicles to their original low
 sensitivity. The above results are interpreted in terms of glycophorin
 acting as a ***receptor*** for HlyA in RBC. The binding constant of
 HlyA for glycophorin was estimated, in RBC at sublytic HlyA
 concentrations, to. . .
 ORGN Classifier
 Enterobacteriaceae 06702
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Organism Name
 Escherichia coli
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 20 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on
 STN
 AN 2001:941959 SCISEARCH <<LOGINID::20091202>>
 GA The Genuine Article (R) Number: 493YA
 TI ***Bacterial*** ***ghosts*** as carrier and targeting systems
 AU Lubitz W (Reprint)
 CS Univ Vienna, Inst Microbiol & Genet, Dr Bohrgasse 9, A-1030 Vienna,
 Austria (Reprint)
 E-mail: werner.lubitz@univie.ac.at
 CS Univ Vienna, Inst Microbiol & Genet, A-1030 Vienna, Austria
 CYA Austria
 SO EXPERT OPINION ON BIOLOGICAL THERAPY, (SEP 2001) Vol. 1, No. 5, pp.
 765-771.
 ISSN: 1471-2598.
 PB INFORMA HEALTHCARE, TELEPHONE HOUSE, 69-77 PAUL STREET, LONDON EC2A 4LQ,
 ENGLAND.
 DT General Review; Journal
 LA English
 REC Reference Count: 48
 ED Entered STN: 7 Dec 2001
 Last Updated on STN: 24 Dec 2008
 ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
 AB ***Bacterial*** ***ghosts*** are empty cell envelopes
 originating from Gram-negative ***bacteria***. They have a natural
 outer surface make-up which provides them with the original targeting
 functions of the ***bacteria*** they are derived from and are thus
 able to bind to and/or are taken up by specific cells or tissues of
 animal, human or plant origin. The extended ***bacterial***
 ghost system represents a platform technology for creating new
 qualities in non-living carriers which can be used for the specific
 targeting of drugs, DNA or other compounds to overcome toxic or
 non-desired obstacles. Freeze dried ***bacterial*** ***ghosts***
 are stable without the requirement of a cold chain and can be effectively
 administered orally and aerogenically as drug carriers. The new system is
 an alternative to liposomes and may have an advantage due to its higher
 specificity for targeting specific tissues, its easy method of production
 and its versatility in entrapping and packaging various compounds in
 different compartments of the carriers.
 TI ***Bacterial*** ***ghosts*** as carrier and targeting systems
 AB ***Bacterial*** ***ghosts*** are empty cell envelopes
 originating from Gram-negative ***bacteria***. They have a natural
 outer surface make-up which provides them with the original targeting
 functions of the ***bacteria*** they are derived from and are thus
 able to bind to and/or are taken up by specific cells or tissues of
 animal, human or plant origin. The extended ***bacterial***
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 non-desired obstacles. Freeze dried ***bacterial*** ***ghosts***
 are stable without the requirement of a cold chain and can be effectively
 administered orally and aerogenically as drug carriers.. . .
 ST Author Keywords: ***bacterial*** ***ghost*** ; carrier and
 targeting systems; drug delivery; synthetic gene delivery; therapy
 STP KeyWords Plus (R): E-MEDIATED LYSIS; PHI-X174 GENE-E; ESCHERICHIA-COLI;
 MANNOSE ***RECEPTOR*** ; PROTEIN-E; ENDOTHELIAL-CELLS; CANDIDATE
 VACCINES; S-LAYERS; DENDRITIC CELLS; IN-SITU

L7 ANSWER 21 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 2002:223200 BIOSIS <<LOGINID::20091202>>

DN PREV200200223200

TI Functional P pilus-specific antibodies that block attachment of
 bacteria to digalactosyl receptors.

AU Jian, L. [Reprint author]; Fusco, P. C. [Reprint author]

CS Baxter Healthcare Corporation, Columbia, MD, USA

SO Abstracts of the General Meeting of the American Society for Microbiology,
 (2001) Vol. 101, pp. 340. print.
 Meeting Info.: 101st General Meeting of the American Society for
 Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society of
 Microbiology.
 ISSN: 1060-2011.

DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 3 Apr 2002
 Last Updated on STN: 3 Apr 2002

AB P pili from uropathogenic Escherichia coli have previously been used to
 formulate vaccines to demonstrate protection against urinary tract
 infections in mouse models. We have previously reported the role of P
 pili in eliciting functionally active antibodies that block attachment of
 purified pili to digalactosyl receptors on human erythrocyte
 ghosts, independent of their tip adhesin, using an inhibition
 ELISA-based method. In this study, an inhibition agglutination assay was
 used to measure the inhibition of the pilated ***bacteria*** binding
 to digalactosyl receptors on both human erythrocyte ***ghosts*** and
 digalactosyl latex beads, using pilus-specific antisera, purified IgG, and
 Fab fragments. Homologous and heterologous inhibition of
 bacterial attachment was demonstrated with rabbit antisera
 against
 F71, F72, F9, and F13 pili. Homologous endpoint inhibition titers for
 digalactosyl latex beads were 12,800-25,600, which were 8-16 times higher
 than with unrelated negative control sera. Heterologous inhibition titers
 were 1-8 times higher than negative controls. ***Bacterial***
 agglutination of human erythrocyte ***ghosts*** was inhibited by 50%
 using homologous F71 antiserum diluted 1:38,400. In order to demonstrate
 direct blocking of ***bacterial*** attachment independent of
 bacterial agglutination, F71-specific Fab fragments were produced
 and were shown to completely inhibit ***bacterial*** agglutination of
 digalactosyl latex beads at 33 mug, with 70% inhibition occurring at 2.1
 mug. In conclusion, evidence of adhesin-independent pilus-specific
 blocking of attachment has been extended in vitro from purified pili to
 pilated ***bacteria***.

TI Functional P pilus-specific antibodies that block attachment of
 bacteria to digalactosyl receptors.

AB. . . of P pili in eliciting functionally active antibodies that block
 attachment of purified pili to digalactosyl receptors on human erythrocyte
 ghosts, independent of their tip adhesin, using an inhibition
 ELISA-based method. In this study, an inhibition agglutination assay was
 used to measure the inhibition of the pilated ***bacteria*** binding
 to digalactosyl receptors on both human erythrocyte ***ghosts*** and
 digalactosyl latex beads, using pilus-specific antisera, purified IgG, and
 Fab fragments. Homologous and heterologous inhibition of
 bacterial attachment was demonstrated with rabbit antisera
 against

F71, F72, F9, and F13 pili. Homologous endpoint inhibition titers for digalactosyl latex. . . were 8-16 times higher than with unrelated negative control sera. Heterologous inhibition titers were 1-8 times higher than negative controls. ***Bacterial*** agglutination of human erythrocyte ***ghosts*** was inhibited by 50% using homologous F71 antiserum diluted 1:38,400. In order to demonstrate direct blocking of ***bacterial*** attachment independent of ***bacterial*** agglutination, F71-specific Fab fragments were produced and were shown to completely inhibit ***bacterial*** agglutination of digalactosyl latex beads at 33 mug, with 70% inhibition occurring at 2.1 mug. In conclusion, evidence of adhesin-independent pilus-specific blocking of attachment has been extended in vitro from purified pili to pilated ***bacteria*** .

IT . . .
 IT Infection
 IT Parts, Structures, & Systems of Organisms
 IT P pilus; erythrocyte: blood and lymphatics
 IT Chemicals & Biochemicals
 IT antibody; digalactosyl ***receptor*** ; immunoglobulin G
 IT Miscellaneous Descriptors
 IT ***bacterial*** agglutination; Meeting Abstract

ORGN Classifier
 Enterobacteriaceae 06702
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Organism Name
 Escherichia coli: pathogen
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human
 Taxa Notes
 Animals, Chordates, . . .

L7 ANSWER 22 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 2000:623585 CAPLUS <<LOGINID::20091202>>
 DN 133:227782
 TI ***Bacterial*** ***ghosts*** as carrier and targeting vehicles
 IN Huter, Veronika; Lubitz, Werner
 PA Austria
 SO Ger. Offen., 10 pp.
 CODEN: GWXXBX
 DT Patent
 LA German
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	----	-----	-----	-----
PI	DE 19909770	A1	20000907	DE 1999-19909770	19990305
	CA 2370714	A1	20000914	CA 2000-2370714	20000303
	WO 2000053163	A1	20000914	WO 2000-EP1906	20000303
	W:			AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,	
				CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,	
				IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,	

MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1158966	A1	20011205	EP 2000-912549	20000303
EP 1158966	B1	20030611		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002538198	T	20021112	JP 2000-603652	20000303
AT 242630	T	20030615	AT 2000-912549	20000303
NZ 514408	A	20040130	NZ 2000-514408	20000303
AU 778166	B2	20041118	AU 2000-34272	20000303
PRAI DE 1999-19909770	A	19990305		
WO 2000-EP1906	W	20000303		

AB Empty ***bacterial*** envelopes (***ghosts***), produced by controlled heterologous expression of a gene which effects a partial lysis of the cell membrane, are useful as carriers and targeting vehicles for active substances and markers. They may be administered via the natural infection pathways for pathogenic ***bacteria*** and are delivered specifically to the target tissues of the ***bacteria*** with high efficiency. Being empty, they can be loaded with active substances to a high degree. Agents which can be packaged in the ***ghosts*** include drugs, polypeptides, nucleic acids, agrochems., dyes, inks, and cosmetics; these may be immobilized by binding to specific receptors or binding sites incorporated into or anchored to the ***ghosts***. Thus, Escherichia coli NM522 cells were transformed simultaneously with plasmid pML1 (contg. phage .phi.X174 gene E encoding a transmembrane protein which induces leakage of the cell contents) and plasmid pAV1 (contg. the 54 5'-terminal codons of gene E fused in-frame to a coding sequence for the protease factor Xa recognition sequence and to 160 codons of the streptavidin gene). Expression of the streptavidin gene was induced with 3 mM IPTG, and expression of lysis protein E was subsequently induced by raising the temp. from 28.degree. to 42.degree.. Centrifugation of the cells and resuspension in distd. water resulted in immediate lysis, producing ***ghosts*** to which streptavidin was anchored. These ***ghosts*** strongly bound biotinylated alk. phosphatase, FITC- ***biotin***, and other biotinylated agents.

OSC.G 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

TI ***Bacterial*** ***ghosts*** as carrier and targeting vehicles

AB Empty ***bacterial*** envelopes (***ghosts***), produced by controlled heterologous expression of a gene which effects a partial lysis of the cell membrane, are useful as. . . carriers and targeting vehicles for active substances and markers. They may be administered via the natural infection pathways for pathogenic ***bacteria*** and are delivered specifically to the target tissues of the ***bacteria*** with high efficiency. Being empty, they can be loaded with active substances to a high degree. Agents which can be packaged in the ***ghosts*** include drugs, polypeptides, nucleic acids, agrochems., dyes, inks, and cosmetics; these may be immobilized by binding to specific receptors or binding sites incorporated into or anchored to the ***ghosts***. Thus, Escherichia coli NM522 cells were transformed simultaneously with plasmid pML1 (contg. phage .phi.X174 gene E encoding a transmembrane protein. . . the temp. from 28.degree. to 42.degree.. Centrifugation of the cells and resuspension in distd. water resulted in immediate lysis, producing ***ghosts*** to which streptavidin was anchored. These ***ghosts*** strongly bound biotinylated alk.

phosphatase, FITC- ***biotin*** , and other biotinylated agents.

ST ***bacteria*** ***ghost*** drug carrier targeting; streptavidin
 bacteria ***ghost*** drug carrier

IT Gene, microbial
 RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (E, of phage .phi.X174, plasmid contg.; ***bacterial***
 ghosts as carrier and targeting vehicles)

IT Polymers, biological studies
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (active agent immobilization in matrix of; ***bacterial***
 ghosts as carrier and targeting vehicles)

IT Diagnosis
 (agents; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Agrochemicals
 Anti-infective agents
 Antitumor agents
 Autoimmune disease
 Bacteria (Eubacteria)
 Cell membrane
 Cytolysis
 Drug targeting
 Dyes
 Gene therapy
 Genetic markers
 Gram-negative ***bacteria***
 Gram-positive ***bacteria*** (Firmicutes)
 Immobilization, biochemical
 Vaccines
 (***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Nucleic acids
 Reporter gene
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Avidins
 Polysaccharides, biological studies
 Receptors
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Drug delivery systems
 (carriers; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT DNA
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
 (fluorescent-labeled; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Coliphage .phi.X174
 (gene E protein of, lysis by; ***bacterial*** ***ghosts*** as carrier and targeting vehicles)

IT Fatty acids, biological studies
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (hydroxy, polymers; ***bacterial*** ***ghosts*** as carrier and
 targeting vehicles)

IT Proteins, specific or class
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (***ligand*** -binding; ***bacterial*** ***ghosts*** as
 carrier and targeting vehicles)

IT Aggregation
 (matrix formation by; ***bacterial*** ***ghosts*** as carrier
 and targeting vehicles)

IT Enzymes, uses
 RL: CAT (Catalyst use); USES (Uses)
 (matrix polymn. catalyzed by; ***bacterial*** ***ghosts*** as
 carrier and targeting vehicles)

IT Encapsulation
 (microencapsulation; ***bacterial*** ***ghosts*** as carrier
 and targeting vehicles)

IT Plasmids
 (streptavidin gene-contg.; ***bacterial*** ***ghosts*** as
 carrier and targeting vehicles)

IT Fusion proteins (chimeric proteins)
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (streptavidin-contg.; ***bacterial*** ***ghosts*** as carrier
 and targeting vehicles)

IT Protamines
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (sulfates; ***bacterial*** ***ghosts*** as carrier and
 targeting vehicles)

IT 146397-20-8
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
 (Biological study); PROC (Process)
 (DNA labeled with; ***bacterial*** ***ghosts*** as carrier and
 targeting vehicles)

IT 25988-63-0, Poly-L-lysine hydrobromide 35013-72-0, ***Biotin***
 N-hydroxysuccinimide ester
 RL: RCT (Reactant); RACT (Reactant or reagent)
 (***bacterial*** ***ghosts*** as carrier and targeting
 vehicles)

IT 9004-54-0, Dextran, biological studies 9013-20-1, Streptavidin
 25104-18-1, Poly-L-lysine 38000-06-5, Poly-L-lysine
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (***bacterial*** ***ghosts*** as carrier and targeting
 vehicles)

IT 9001-78-9D, biotinylated 25104-18-1D, Poly-L-lysine, biotinylated
 38000-06-5D, Poly-L-lysine, biotinylated 134759-22-1
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
 (Biological study); PROC (Process)
 (binding of, to streptavidin-contg. ***bacterial*** ***ghosts***
 ; ***bacterial*** ***ghosts*** as carrier and targeting
 vehicles)

L7 ANSWER 23 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on
 STN

AN 2000:710892 SCISEARCH <<LOGINID::20091202>>

GA The Genuine Article (R) Number: 354GJ

TI Interaction of Bartonella bacilliformis with human erythrocyte membrane

proteins

AU Hill E M (Reprint)

CS Meharry Med Coll, Sch Grad Studies, Dept Microbiol, Nashville, TN 37208
USA (Reprint)

AU Buckles E L

CS Meharry Med Coll, Sch Med, Nashville, TN 37208 USA

CYA USA

SO MICROBIAL PATHOGENESIS, (SEP 2000) Vol. 29, No. 3, pp. 165-174.
ISSN: 0882-4010.

PB ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.

DT Article; Journal

LA English

REC Reference Count: 25

ED Entered STN: 2000
Last Updated on STN: 2000
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Intracellular invasion is an important aspect of Carrion's disease caused by *Bartonella bacilliformis*. Both the hematic and tissue phases of the disease involve the initial attachment of the organism to erythrocytes and endothelial cells, respectively. Using two different approaches, preliminary evidence is provided that *B. bacilliformis* interacts with multiple surface-exposed proteins on human erythrocytes. Utilizing Western blot analysis, it was demonstrated that the organism binds several biotinylated erythrocyte proteins with approximate molecular masses of 230, 210, 100, 83 and 44 kDa. There was enhanced *Bartonella* binding to the 44 kDa protein and binding to a 25 kDa protein following exposure of intact red cells to trypsin. Moreover, there was a complete abrogation of binding to these proteins following exposure of erythrocytes to sodium metaperiodate oxidation, indicating the significance of carbohydrate moieties in the interactions of *Bartonella* with the erythrocyte. In a second approach, similar binding proteins or putative receptors were identified when *Bartonella* was cc-incubated with isolated membrane proteins from red cell ***ghosts***. A comparison of the molecular weights of these putative receptors with known erythrocyte proteins and their immunoreactivity to specific antisera suggested that the 230 and 210 kDa proteins are the alpha and beta subunits of spectrin; the 100 and 83 kDa proteins are band 3 protein and glycophorin A, respectively; and the 44 and 25 kDa proteins are the respective dimeric and monomeric forms of glycophorin B. Consistent with this notion was the binding of *Bartonella* to purified preparations of alpha and beta spectrin and glycophorin A/B. (C) 2000 Academic Press.

AB . . . approach, similar binding proteins or putative receptors were identified when *Bartonella* was cc-incubated with isolated membrane proteins from red cell ***ghosts***. A comparison of the molecular weights of these putative receptors with known erythrocyte proteins and their immunoreactivity to specific antisera. . . .

ST Author Keywords: *Bartonella bacilliformis*; ***bacterial*** adherence; human erythrocytes; glycophorin; band 3 protein; spectrin

STP KeyWords Plus (R): PLASMODIUM-FALCIPARUM; ENDOTHELIAL-CELLS; INVASION; HENSELAE; ***RECEPTOR***; ENTRY

L7 ANSWER 24 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

AN 2000307384 EMBASE <<LOGINID::20091202>>

TI Characterization of p40/GPR69A as a peripheral membrane protein related to the lantibiotic synthetase component C.

AU Bauer, Hemma; Mayer, Herbert; Salzer, Ulrich; Prohaska, Rainer

(correspondence)

CS Institute of Medical Biochemistry, Department of Biochemistry, University of Vienna, Dr. Bohr-Gasse 9/3, A-1030 Vienna, Austria. prohaska@bch.univie.ac.at

AU Marchler-Bauer, Aron

CS National Center for Biotechnology Information, National Institutes of Health, Bethesda, MD 20894, United States.

SO Biochemical and Biophysical Research Communications, (18 Aug 2000) Vol. 275, No. 1, pp. 69-74.

Refs: 19

ISSN: 0006-291X CODEN: BBRCA9

CY United States

DT Journal; Article

FS 029 Clinical and Experimental Biochemistry

LA English

SL English

ED Entered STN: 21 Sep 2000

Last Updated on STN: 21 Sep 2000

AB The 40 kDa erythrocyte membrane protein p40/GPR69A, previously assigned to the G-protein-coupled ***receptor*** superfamily, was now identified by peptideantibodies and characterized as a loosely associated peripheral membrane protein. This result is in striking contrast to the proposed seven-transmembrane protein structure and function and therefore we wish to correct our previous proposal. p40 is located at the cytoplasmic side of the membrane and is neither associated with the cytoskeleton nor lipid rafts. Refined sequence analysis revealed that p40 is related to the LanC family of ***bacterial*** membrane-associated proteins which are involved in the biosynthesis of antimicrobial peptides. Therefore, we rename p40 to LanC-like protein 1 (LANCL1) and suggest that it may play a similar role as a peptide-modifying enzyme component in eukaryotic cells. (C) 2000 Academic Press.

AB The 40 kDa erythrocyte membrane protein p40/GPR69A, previously assigned to the G-protein-coupled ***receptor*** superfamily, was now identified by peptideantibodies and characterized as a loosely associated peripheral membrane protein. This result is in striking. . . associated with the cytoskeleton nor lipid rafts. Refined sequence analysis revealed that p40 is related to the LanC family of ***bacterial*** membrane-associated proteins which are involved in the biosynthesis of antimicrobial peptides. Therefore, we rename p40 to LanC-like protein 1 (LANCL1). . .

CT Medical Descriptors:

amino acid sequence

article

erythrocyte ghost

*erythrocyte membrane

human

human cell

immunochemistry

priority journal

*protein analysis

protein degradation

protein structure

****G protein coupled receptor***

*lanthionine

*lantibiotic

*membrane protein

*peptide antibody

*synthetase

L7 ANSWER 25 OF 68 MEDLINE on STN
 AN 1999456571 MEDLINE <<LOGINID::20091202>>
 DN PubMed ID: 10525277
 TI Maitotoxin-induced calcium influx in erythrocyte ***ghosts*** and rat glioma C6 cells, and blockade by gangliosides and other membrane lipids.
 AU Konoki K; Hashimoto M; Murata M; Tachibana K
 CS Department of Chemistry, School of Science, The University of Tokyo, Hongo, Bunkyo-ku, Tokyo 113-0033, Japan.
 SO Chemical research in toxicology, (1999 Oct) Vol. 12, No. 10, pp. 993-1001. Journal code: 8807448. ISSN: 0893-228X.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS Priority Journals
 EM 200001
 ED Entered STN: 14 Jan 2000
 Last Updated on STN: 14 Jan 2000
 Entered Medline: 4 Jan 2000
 AB Maitotoxin (MTX) at 0.3 nM elicited a 10-20-fold increase in the level of Ca(2+) influx in rat glioma C6 cells. At higher doses (3-30 nM), MTX induced marked Ca(2+) influx in human erythrocyte ***ghosts*** when monitored with the fluorescent dye Fura-2. Although the ***ghosts*** were not as susceptible to MTX as intact erythrocytes or other cell lines, Fura-2 experiments under various conditions suggested that the MTX-induced entry of ions into the ***ghosts*** was mediated by a mechanism similar to that reported for cells or tissues. These ***ghosts*** are the simplest system known to be sensitive to MTX and thus may be suitable for research on the direct action of MTX. Gangliosides GM1 and GM3, glycosphingolipids which have a sialic acid residue, strongly inhibited MTX-induced Ca(2+) influx in C6 cells, while the inhibitory action by asialo-GM1, which lacks a sialic acid residue, was somewhat weaker. Their inhibitory potencies were in the following order: GM1 (IC(50) approximately 2 microM) > GM3 (IC(50) approximately 5 microM) > asialo-GM1 (IC(50) approximately 20 microM). GM1 (3 microM) completely blocked MTX (30 nM)-induced Ca(2+) influx in human erythrocyte ***ghosts***. When C6 cells were pretreated with tunicamycin, an antibiotic which inhibits N-linked glycosylation, or concanavalin A, a ***lectin*** which exhibits a high affinity for cell-surface oligosaccharides, MTX-induced Ca(2+) influx was significantly potentiated. This suggests that removal of oligosaccharides from the cell surface by tunicamycin or capping of sugar chains on plasma membranes by concanavalin A can potentiate the action of MTX.
 TI Maitotoxin-induced calcium influx in erythrocyte ***ghosts*** and rat glioma C6 cells, and blockade by gangliosides and other membrane lipids.
 AB . . . Ca(2+) influx in rat glioma C6 cells. At higher doses (3-30 nM), MTX induced marked Ca(2+) influx in human erythrocyte ***ghosts*** when monitored with the fluorescent dye Fura-2. Although the ***ghosts*** were not as susceptible to MTX as intact erythrocytes or other cell lines, Fura-2 experiments under various conditions suggested that the MTX-induced entry of ions into the ***ghosts*** was mediated by a mechanism similar to that reported for cells or tissues. These ***ghosts*** are the simplest system known to be sensitive to MTX and thus may be suitable for research on the direct. . . microM) > asialo-GM1 (IC(50) approximately 20 microM). GM1 (3 microM) completely blocked MTX (30 nM)-induced Ca(2+) influx in human erythrocyte

ghosts . When C6 cells were pretreated with tunicamycin, an antibiotic which inhibits N-linked glycosylation, or concanavalin A, a ***lectin*** which exhibits a high affinity for cell-surface oligosaccharides, MTX-induced Ca(2+) influx was significantly potentiated. This suggests that removal of oligosaccharides. . .

CT Animals

*** Anti-Bacterial Agents: PD, pharmacology***

*Brain Neoplasms: ME, metabolism

*Calcium: BL, blood

Calcium Radioisotopes: DU, diagnostic use

Concanavalin A: PD, pharmacology

CN 0 (Anti- ***Bacterial*** Agents); 0 (Calcium Radioisotopes); 0 (Fluorescent Dyes); 0 (Gangliosides); 0 (Marine Toxins); 0 (Membrane Lipids); 0 (Oxocins)

L7 ANSWER 26 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1999:828113 SCISEARCH <<LOGINID::20091202>>

GA The Genuine Article (R) Number: 250PR

TI ***Bacterial*** cell envelopes (***ghosts***) but not S-layers activate human endothelial cells (HUVECs) through sCD14 and LBP mechanism

AU Furst-Ladani S (Reprint)

CS Ludwig Boltzmann Inst Expt & Clin Traumatol, Donaueschingenstr 13, A-1200 Vienna, Austria (Reprint)

AU Redl H; Haslberger A; Lubitz W; Messner P; Sleytr U B; Schlag G

CS Ludwig Boltzmann Inst Expt & Clin Traumatol, A-1200 Vienna, Austria; Univ Vienna, Inst Microbiol & Genet, A-1090 Vienna, Austria; Univ Agr Sci, Ctr Ultrastruct Res, Vienna, Austria; Univ Agr Sci, Ludwig Boltzmann Inst Mol Nanotechnol, Vienna, Austria

CYA Austria

SO VACCINE, (14 OCT 1999) Vol. 18, No. 5-6, pp. 440-448. ISSN: 0264-410X.

PB ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND.

DT Article; Journal

LA English

REC Reference Count: 26

ED Entered STN: 1999

Last Updated on STN: 1999

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB ***Bacterial*** cell-envelopes (called ***ghosts***) and surface layers (S-layers) are discussed to be used as vaccines and/or adjuvants, consequently it is necessary to find out which immunomodulatory mediators are induced in human cells. The present work focuses on the effects of ***ghosts*** (Escherichia coli O26:B6), S-layers (Bacillus stearothermophilus) in comparison with LPS and antibiotic-inactivated whole ***bacteria*** (E. coli O26:B6) on human umbilical vein endothelial cells (HUVEC) with regard to the release of interleukin 6 (IL-6) and the expression of surface E-selectin and the role of lipopolysaccharide binding protein (LBP), soluble CD14 (sCD14) and serum for this activation.

Endothelial cells responded to ***ghosts*** , whole ***bacteria*** and LPS with IL-6 release up to 15000 pg/ml and surface E-selectin expression, while in contrast the response to S-layers with IL-6 release up to 500 pg/ml was very weak. Compared to LPS, 10-100-fold higher concentrations of ***bacterial*** ***ghosts*** and whole

bacteria were required to induce the cytokine synthesis and E-selectin expression. IL-6 release and E-selectin expression of HUVECs were reduced in the absence of serum and equivalent to unstimulated samples. We have also studied the role of CD14 and LBP for the activation of endothelial cells using antiCD14 and antiLBP antibodies (Ab). AntiCD14 and antiLBP Ab both inhibited IL-6 release and E-selectin expression in a dose dependent manner after stimulation with ***ghosts***, whole ***bacteria*** and LPS but had no effect on S-layers stimulated cells. AntiCD14 Ab inhibited more effectively than antiLBP Ab. These findings suggest that ***bacterial*** ***ghosts*** but not: S-layers activate HUVECs through sCD14 and LBP dependent mechanisms. (C) 1999 Elsevier Science Ltd. All rights reserved.

TI ***Bacterial*** cell envelopes (***ghosts***) but not S-layers activate human endothelial cells (HUVECs) through sCD14 and LBP mechanism

AB ***Bacterial*** cell-envelopes (called ***ghosts***) and surface layers (S-layers) are discussed to be used as vaccines and/or adjuvants, consequently it is necessary to find out which immunomodulatory mediators are induced in human cells. The present work focuses on the effects of ***ghosts*** (Escherichia coli O26:B6), S-layers (Bacillus stearothermophilus) in comparison with LPS and antibiotic-inactivated whole ***bacteria*** (E. coli O26:B6) on human umbilical vein endothelial cells (HUVEC) with regard to the release of interleukin 6 (IL-6) and. . . and the role of lipopolysaccharide binding protein (LBP), soluble CD14 (sCD14) and serum for this activation.

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ST Author Keywords: endotoxicity; ***bacterial*** ***ghosts*** ; S-layers; cytokines; HUVEC

STP KeyWords Plus (R): LIPOPOLYSACCHARIDE-BINDING-PROTEIN; SOLUBLE CD14; IN-VITRO; LPS; MACROPHAGES; INDUCTION; RESPONSES; ENDOTOXIN; ALLERGEN; ***RECEPTOR***

L7 ANSWER 27 OF 68 MEDLINE on STN

AN 1999411196 MEDLINE <<LOGINID::20091202>>

DN PubMed ID: 10481582

TI [Binding of the antileukemia drug Escherichia coli L-asparaginase to the plasma membrane of normal human mononuclear cells].
Asociacion de la droga antileucemica L-asparaginasa de Escherichia coli a la membrana plasmatica de celulas mononucleares humanas normales.

AU Mercado-Vianco L; Arenas-Diaz G

CS Laboratorio de Fisiologia Celular, Instituto de Biologia, Universidad Catolica de Valparaiso, Chile.

SO Sangre, (1999 Jun) Vol. 44, No. 3, pp. 204-9.
Journal code: 0404373. ISSN: 0036-4355.

CY Spain
 DT (ENGLISH ABSTRACT)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA Spanish
 FS Priority Journals
 EM 199910
 ED Entered STN: 14 Oct 1999
 Last Updated on STN: 14 Oct 1999
 Entered Medline: 7 Oct 1999
 AB OBJECTIVE: To demonstrate that the enzyme L-asparaginase from Escherichia coli (EcA) binds to the plasma membranes of normal human lymphocytes and monocytes. MATERIAL AND METHODS: Lymphocytes and monocytes were isolated from heparinized blood samples which came from healthy volunteer donors. The cells were incubated with EcA to detect a possible binding of the enzyme to the mononuclear cells by indirect immunofluorescence using confocal microscopy. Meanwhile, ultracentrifugation was used to obtain the erythrocyte ***ghost*** microsomal fraction (P100) which was then analyzed by Western blotting to determine if EcA binds the lipid bilayer unspecifically. For the immunoassays, monospecific polyclonal antibodies were obtained from ascitic tumors developed in mice immunized with commercial L-asparaginase. RESULTS: EcA binds the lymphocyte and monocyte plasma membranes. In monocytes, there occurs a capping phenomenon, that is, the accumulation of fluorescent marker in one region. The image analyzer highlights it clearly at a depth of 3.8 microns. This binding would be unspecific, that is, there is no mediation of a specific ***receptor*** that binds EcA. This arises from the ability of the enzyme to bind to the membranes of erythrocyte ***ghost***, as evidenced by the ability of the molecule to associate with a hydrophobic medium. The antibodies against EcA obtained from ascitic tumours developed in mice do not show cross reactivity with Na+/K+ ATPase, aspartate aminotransferase, nor with extracts of blood cells, which would make it a specific tool for the detection of EcA in whole cells and in homogenates electrotransferred to nitrocellulose membranes. CONCLUSION: L-asparaginase from E. coli behaves as a lipoprotein due to its ability to insert itself into hydrophobic environments, in which it resembles an isozyme present in T. pyriformis. The binding of this enzyme to lymphocytes and monocytes, demonstrated in this work, would permit the modification of the antileukemic treatment injecting doses of EcA bound to patient's own isolated immune cells.
 AB . . . the enzyme to the mononuclear cells by indirect immunofluorescence using confocal microscopy. Meanwhile, ultracentrifugation was used to obtain the erythrocyte ***ghost*** microsomal fraction (P100) which was then analyzed by Western blotting to determine if EcA binds the lipid bilayer unspecifically. For. . . at a depth of 3.8 microns. This binding would be unspecific, that is, there is no mediation of a specific ***receptor*** that binds EcA. This arises from the ability of the enzyme to bind to the membranes of erythrocyte ***ghost***, as evidenced by the ability of the molecule to associate with a hydrophobic medium. The antibodies against EcA obtained from. . .
 CT . . . Agents: AD, administration & dosage
 *Antineoplastic Agents: ME, metabolism
 Ascites: IM, immunology
 Asparaginase: AD, administration & dosage
 *Asparaginase: ME, metabolism
 *** Bacterial Proteins: AD, administration & dosage***

Bacterial Proteins: ME, metabolism
 *Cell Membrane: ME, metabolism
 Cross Reactions
 Drug Carriers
 Erythrocyte Membrane: ME, metabolism
 Escherichia coli: EN, enzymology
 Fluorescent. . . DE, drug effects
 Lymphocytes: ME, metabolism
 Membrane Lipids: ME, metabolism
 Mice
 Microscopy, Confocal
 Monocytes: DE, drug effects
 Monocytes: ME, metabolism
 *** Receptor Aggregation***
 Thromboembolism: CI, chemically induced
 Thromboembolism: PC, prevention & control
 CN 0 (Antibodies, Monoclonal); 0 (Antineoplastic Agents); 0 (
 Bacterial Proteins); 0 (Drug Carriers); 0 (Membrane Lipids); EC
 3.5.1.1 (Asparaginase)

 L7 ANSWER 28 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 1999:557792 CAPLUS <<LOGINID::20091202>>
 DN 131:355959
 TI ***Bacterial*** ***ghosts*** as drug carrier and targeting
 vehicles
 AU Huter, V.; Szostak, M. P.; Gampfer, J.; Prethaler, S.; Wanner, G.; Gabor,
 F.; Lubitz, W.
 CS Institute of Microbiology and Genetics, University of Vienna, Vienna,
 A-1030, Austria
 SO Journal of Controlled Release (1999), 61(1-2), 51-63
 CODEN: JCREEC; ISSN: 0168-3659
 PB Elsevier Science Ireland Ltd.
 DT Journal
 LA English
 AB A novel system for the packaging of drugs as well as vaccines is
 presented. ***Bacterial*** ***ghosts*** are intact, non-denatured
 bacterial envelopes that are created by lysis of ***bacteria***
 through the expression of cloned phage PhiX174 gene E. Inhibition of
 induced E-mediated lysis by MgSO₄, harvesting of cells by centrifugation,
 and resuspension in low-ionic-strength buffers leads to rapid, violent
 lysis and results in empty ***bacterial*** envelopes with large
 (approx. 1 .mu.m in diam.) openings. The construction of plasmid pAV1,
 which encodes a streptavidin fusion protein with an N-terminal membrane
 anchor sequence, allows the loading of the inner side of the cytoplasmic
 membrane with streptavidin. The functionality and efficacy of binding of
 even large biotinylated compds. in such streptavidin ***ghosts*** (SA-
 ghosts) was assessed using the enzyme alk. phosphatase. The
 successful binding of biotinylated fluorescent dextran, as well as
 fluorescent DNA complexed with biotinylated polylysine, was demonstrated
 microscopically. The display by ***bacterial*** ***ghosts*** of
 morphol. and antigenic surface structures of their living counterparts
 permits their attachment to target tissues such as the mucosal surfaces of
 the gastrointestinal and respiratory tract, and their uptake by phagocytes
 and M cells. In consequence, SA- ***ghosts*** are proposed as drug
 carriers for site-specific drug delivery.
 OSC.G 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)
 RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

- TI ***Bacterial*** ***ghosts*** as drug carrier and targeting vehicles
- AB A novel system for the packaging of drugs as well as vaccines is presented. ***Bacterial*** ***ghosts*** are intact, non-denatured ***bacterial*** envelopes that are created by lysis of ***bacteria*** through the expression of cloned phage PhiX174 gene E. Inhibition of induced E-mediated lysis by MgSO₄, harvesting of cells by centrifugation, and resuspension in low-ionic-strength buffers leads to rapid, violent lysis and results in empty ***bacterial*** envelopes with large (approx. 1 .mu.m in diam.) openings. The construction of plasmid pAV1, which encodes a streptavidin fusion protein. . . of the cytoplasmic membrane with streptavidin. The functionality and efficacy of binding of even large biotinylated compds. in such streptavidin ***ghosts*** (SA-***ghosts***) was assessed using the enzyme alk. phosphatase. The successful binding of biotinylated fluorescent dextran, as well as fluorescent DNA complexed with biotinylated polylysine, was demonstrated microscopically. The display by ***bacterial*** ***ghosts*** of morphol. and antigenic surface structures of their living counterparts permits their attachment to target tissues such as the mucosal surfaces of the gastrointestinal and respiratory tract, and their uptake by phagocytes and M cells. In consequence, SA- ***ghosts*** are proposed as drug carriers for site-specific drug delivery.
- ST ***bacterial*** ***ghost*** drug delivery vehicle targeting
- IT ***Bacteria*** (Eubacteria)
Drug targeting
Genetic vectors
Phagocytosis
(***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- IT Drug delivery systems
(***bacterial*** ***ghosts*** ; ***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- IT Cell membrane
(streptavidin-modified; ***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- IT Biological transport
(uptake, ***receptor*** -mediated; ***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- IT 9013-20-1D, Streptavidin, membrane conjugates
RL: BPR (Biological process); BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); PROC (Process); RACT (Reactant or reagent)
(***bacterial*** ***ghosts*** as drug carrier and targeting vehicles)
- L7 ANSWER 29 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN
- AN 1998:503225 SCISEARCH <<LOGINID::20091202>>
- GA The Genuine Article (R) Number: ZX307
- TI ***Bacterial*** cell envelopes (***ghosts***) and LPS but not ***bacterial*** S-layers induce synthesis of immune-mediators in mouse macrophages involving CD14
- AU Haslberger A G (Reprint)
- CS Univ Vienna, Bioctr, Inst Microbiol & Genet, Dr Bohrgasse 9, A-1030 Vienna, Austria (Reprint)
- AU Mader H J; Schmalnauer M; Kohl G; Szostak M P; Messner P; Sleytr U B;

Wanner G; Furst-Ladani S; Lubitz W

CS Univ Vienna, Bioctr, Inst Microbiol & Genet, A-1030 Vienna, Austria; Agr Univ Vienna, Zentrum Ultrastrukturforsch, A-1180 Vienna, Austria; Agr Univ Vienna, Ludwig Boltzmann Inst Mol Nanotechnol, A-1180 Vienna, Austria; Univ Munich, Inst Bot, D-8000 Munich, Germany; Lorenz Bohler Krankenhaus, Ludwig Boltzmann Inst Expt & Klin Traumatol, Vienna, Austria

CYA Austria; Germany

SO JOURNAL OF ENDOTOXIN RESEARCH, (DEC 1997) Vol. 4, No. 6, pp. 431-441. ISSN: 0968-0519.

PB W S MANEY & SONS LTD, HUDSON RD, LEEDS LS9 7DL, ENGLAND.

DT Article; Journal

LA English

REC Reference Count: 46

ED Entered STN: 1998

Last Updated on STN: 1998

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The synthesis of inflammatory mediators in human macrophages/monocytes seen after stimulation with lipopolysaccharide (LPS) involves the binding of CD14 to LPS complexed to lipopolysaccharide binding protein (LBP). The binding mechanisms of different LPS domains to LBP and CD14, as well as the interaction of the entire ***bacterial*** cell wall and its components with CD14 and LBP, are poorly understood. We, therefore, studied the effects of anti-mouse CD14 antibodies on the synthesis of TNF alpha and PGE(2) in RAW 264.7 mouse macrophages stimulated by ***bacterial*** cell envelopes (***ghosts***) of Escherichia coli O26:B6 and Salmonella typhimurium C5, LPS, lipid A, and crystalline ***bacterial*** cell surface layer (S-layer) preparations. ***Ghosts*** and S-layers, with distinct activities on the immune-system, are presently under investigation for their use as vaccines. Whereas LPS and E. coli ***ghosts*** exhibited a strong endotoxic activity in the Limulus amoebocyte lysate assay, the endotoxic activity of S-layer preparations was several orders of magnitude lower. LPS, ***ghosts***, and ***bacterial*** S-layers all induced TNF alpha and PGE(2) synthesis as well as the accumulation of TNF alpha mRNA. Pre-incubation with anti-mouse CD14 antibodies resulted in a dose-dependent inhibition of TNF alpha and PGE(2) synthesis after stimulation by LPS, lipid A (30-50%) and ***ghosts*** (40-70%). The ***bacterial*** S-layer-induced mediator synthesis remained unchanged following the addition of anti-mouse CD14 antibodies. Reproducible differences could be observed for the inhibition of TNF alpha induced by LPS of different species by anti-CD14. Adding fetal calf serum (FCS) strongly enhanced the release of cell mediators stimulated by low doses of LPS and ***bacterial*** ***ghosts***. These effects of the FCS may be due to the presence of LBP in the FCS. The results show that CD14 is highly relevant for the activation of mouse macrophages by ***bacterial*** cells, LPS, and lipid A. Specially defined ***bacterial*** cell wall constituents such as ***bacterial*** S-layers might act through other activation pathways.

TI ***Bacterial*** cell envelopes (***ghosts***) and LPS but not ***bacterial*** S-layers induce synthesis of immune-mediators in mouse macrophages involving CD14

AB . . . (LBP). The binding mechanisms of different LPS domains to LBP and CD14, as well as the interaction of the entire ***bacterial*** cell wall and its components with CD14 and LBP, are poorly understood. We, therefore, studied the effects of anti-mouse CD14 antibodies on the synthesis of TNF alpha and PGE(2) in RAW 264.7 mouse macrophages stimulated by ***bacterial*** cell envelopes (***ghosts***) of

Escherichia coli O26:B6 and Salmonella typhimurium C5, LPS, lipid A, and crystalline ***bacterial*** cell surface layer (S-layer) preparations. ***Ghosts*** and S-layers, with distinct activities on the immune-system, are presently under investigation for their use as vaccines. Whereas LPS and E. coli ***ghosts*** exhibited a strong endotoxic activity in the Limulus amoebocyte lysate assay, the endotoxic activity of S-layer preparations was several orders of magnitude lower. LPS, ***ghosts***, and ***bacterial*** S-layers all induced TNF alpha and PGE(2) synthesis as well as the accumulation of TNF alpha mRNA. Pre-incubation with anti-mouse. . . antibodies resulted in a dose-dependent inhibition of TNF alpha and PGE(2) synthesis after stimulation by LPS, lipid A (30-50%) and ***ghosts*** (40-70%), The ***bacterial*** S-layer-induced mediator synthesis remained unchanged following the addition of anti-mouse CD14 antibodies. Reproducible differences could be observed for the inhibition. . . anti-CD14. Adding fetal calf serum (FCS) strongly enhanced the release of cell mediators stimulated by low doses of LPS and ***bacterial*** ***ghosts***. These effects of the FCS may be due to the presence of LBP in the FCS, The results show that CD14 is highly relevant for the activation of mouse macrophages by ***bacterial*** cells, LPS, and lipid A. Specially defined ***bacterial*** cell wall constituents such as ***bacterial*** S-layers might act through other activation pathways.

STP KeyWords Plus (R): TUMOR-NECROSIS-FACTOR; FACTOR-ALPHA GENE; BINDING-PROTEIN; TYROSINE PHOSPHORYLATION; LIPOPOLYSACCHARIDE LPS; MURINE MACROPHAGES; ESCHERICHIA-COLI; SURFACE-LAYERS; KAPPA-B; ***RECEPTOR***

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STN DUPLICATE 5

AN 1996:262954 BIOSIS <<LOGINID::20091202>>

DN PREV199698819083

TI Macrophage dysfunction following the phagocytosis of IgG-coated erythrocytes: Production of lipid peroxidation products.

AU Loegering, Daniel J. [Reprint author]; Raley, Michael J.; Reho, Thomas A.; Eaton, John W.

CS Dep. Physiol. Cell Biol., A-134, Albany Med. Coll., 47 New Scotland Ave., Albany, NY 12208-3479, USA

SO Journal of Leukocyte Biology, (1996) Vol. 59, No. 3, pp. 357-362.
CODEN: JLBIE7. ISSN: 0741-5400.

DT Article

LA English

ED Entered STN: 10 Jun 1996

Last Updated on STN: 10 Jun 1996

AB The phagocytosis of erythrocytes may contribute to the increased susceptibility to life-threatening infections in patients with burn injury, sickle cell anemia, and malaria. The phagocytosis of immunoglobulin G-coated erythrocytes (EIgG) is followed by a transient depression of several macrophage functions including phagocytosis, respiratory burst capacity, and killing of ***bacteria***. The present study suggests the possibility that after erythrophagocytosis hemoglobin-derived iron conspires with reactive oxygen products of the macrophage respiratory burst to cause oxidant damage to the phagocyte. Challenge of elicited peritoneal macrophages with EIgG phagocytosis was followed by an increase in lipid peroxidation as assessed by thiobarbituric acid-reactive substances (TBARS). Doses of EIgG associated with increased TBARS also caused a depression of Fc ***receptor***-mediated phagocytosis and phorbol myristate acetate (PMA)-stimulated

hydrogen peroxide production. Time course experiments demonstrated that the increase in TBARS coincided with the depression of macrophage function. There was no increase in TBARS following the phagocytosis of IgG-coated erythrocyte ***ghosts***, suggesting that hemoglobin iron is involved in the generation of TBARS. The phagocytosis of erythrocyte ***ghosts*** did not depress macrophage function. Since complement ***receptor***-mediated phagocytosis does not stimulate the respiratory burst, the role of the respiratory burst in causing lipid peroxidation was assessed using the phagocytosis of complement-coated erythrocytes. Phagocytic challenge with complement-coated erythrocytes caused neither an increase in TBARS nor a depression of macrophage function. However, there was an increase in TBARS when the respiratory burst was stimulated with PMA following complement ***receptor***-mediated phagocytosis of erythrocytes. These results suggest that hemoglobin iron and phagocyte-generated oxidants collaborate to cause the depression of macrophage function following EIgG phagocytosis.

AB. . . erythrocytes (EIgG) is followed by a transient depression of several macrophage functions including phagocytosis, respiratory burst capacity, and killing of ***bacteria***. The present study suggests the possibility that after erythrophagocytosis hemoglobin-derived iron conspires with reactive oxygen products of the macrophage respiratory. . . as assessed by thiobarbituric acid-reactive substances (TBARS). Doses of EIgG associated with increased TBARS also caused a depression of Fc ***receptor***-mediated phagocytosis and phorbol myristate acetate (PMA)-stimulated hydrogen peroxide production. Time course experiments demonstrated that the increase in TBARS coincided with the depression of macrophage function. There was no increase in TBARS following the phagocytosis of IgG-coated erythrocyte ***ghosts***, suggesting that hemoglobin iron is involved in the generation of TBARS. The phagocytosis of erythrocyte ***ghosts*** did not depress macrophage function. Since complement ***receptor***-mediated phagocytosis does not stimulate the respiratory burst, the role of the respiratory burst in causing lipid peroxidation was assessed using. . . of macrophage function. However, there was an increase in TBARS when the respiratory burst was stimulated with PMA following complement ***receptor***-mediated phagocytosis of erythrocytes. These results suggest that hemoglobin iron and phagocyte-generated oxidants collaborate to cause the depression of macrophage function. . .

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STN DUPLICATE 6

AN 1994:227425 BIOSIS <<LOGINID::20091202>>

DN PREV199497240425

TI Pore-formation by Escherichia coli hemolysin (HlyA) and other members of
the RTX toxins family.

AU Menestrina, Gianfranco [Reprint author]; Moser, Claudio; Pellet,
Shahaireen; Welch, Rodney

CS CNR Centro di Fisica degli Stati Aggregati, via Sommarive 14, I-38050
Povo, Trento, Italy

SO Toxicology, (1994) Vol. 87, No. 1-3, pp. 249-267.
CODEN: TXCYAC. ISSN: 0300-483X.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 24 May 1994
Last Updated on STN: 24 May 1994

AB Escherichia coli hemolysin (HlyA) is a major cause of E. coli virulence.

It lyses erythrocytes by a colloid osmotic shock due to the formation of hydrophilic pores in the cell wall. The size of these channels can be estimated using osmotic protectant of increasing dimensions. To show that the formation of pores does not depend critically on the osmotic swelling we prepared resealed human erythrocyte ***ghosts*** loaded with a fluorescent marker. When attacked by HlyA the internal marker was released, indicating the formation of toxin channels so large as to let it through. The channels can be directly demonstrated also in purely lipidic model systems such as planar membranes and unilamellar vesicles, which lack any putative protein ***receptor***. HlyA has been recognised as a member of a large family of exotoxins elaborated by Gram-negative organisms including *Proteus*, *Bordetella*, *Morganella*, *Pasteurella* and *Actinobacillus*. These toxins have quite different target cell specificity and in many cases are leukocidal. When tried on planar membranes however, even specific leukotoxins open channels not dissimilar from those formed by HlyA, suggesting this might be a common step in their action. Comparison of the hydrophobic properties of six members of the toxin family indicates the presence of a conserved cluster of ten contiguous amphipathic helices, located in the N-terminal half of the molecule, which might be involved in channel formation.

AB. . . To show that the formation of pores does not depend critically on the osmotic swelling we prepared resealed human erythrocyte ***ghosts*** loaded with a fluorescent marker. When attacked by HlyA the internal marker was released, indicating the formation of toxin channels. . . directly demonstrated also in purely lipidic model systems such as planar membranes and unilamellar vesicles, which lack any putative protein ***receptor***. HlyA has been recognised as a member of a large family of exotoxins elaborated by Gram-negative organisms including *Proteus*, *Bordetella*, . . .

ORGN Classifier

Enterobacteriaceae 06702

Super Taxa

Facultatively Anaerobic Gram-Negative Rods; Eubacteria;

Bacteria ; Microorganisms

Organism Name

Escherichia coli

Taxa Notes

Bacteria , Eubacteria, Microorganisms

L7 ANSWER 32 OF 68 SCISEARCH COPYRIGHT (c) 2009 The Thomson Corporation on STN
 AN 1993:12050 SCISEARCH <<LOGINID::20091202>>
 GA The Genuine Article (R) Number: KE757
 TI DEPRESSION OF MACROPHAGE RESPIRATORY BURST CAPACITY AND ARACHIDONIC-ACID RELEASE AFTER FC ***RECEPTOR*** -MEDIATED PHAGOCYTOSIS
 AU SCHWACHA M G (Reprint); GUDEWICZ P W; SNYDER J A; LOEGERING D J
 CS UNION UNIV, DEPT PHYSIOL & CELL BIOL, ALBANY, NY 12208
 CYA USA
 SO JOURNAL OF IMMUNOLOGY, (1 JAN 1993) Vol. 150, No. 1, pp. 236-245. ISSN: 0022-1767.
 PB AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
 DT Article; Journal
 FS LIFE
 LA English
 REC Reference Count: 48
 ED Entered STN: 1994
 Last Updated on STN: 1994

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The phagocytosis of IgG-coated erythrocytes (EIgG) by macrophages results in a subsequent depression of macrophage phagocytic function, respiratory burst capacity, and ***bactericidal*** activity. Our study was carried out to determine the importance of impaired arachidonic acid release in the depression of the respiratory burst after EIgG phagocytosis. The depression of triggered H2O2 production after EIgG phagocytosis was not due to cyclooxygenase products because indomethacin or aspirin did not modify the depression. Further studies revealed that the depression of triggered H2O2 production after EIgG phagocytosis was associated with a depression in the ability of macrophages to release arachidonic acid in response to PMA, zymosan, or calcium ionophore. The addition of exogenous arachidonic acid partially prevented the depression of triggered H2O2 production after EIgG phagocytosis. Unlike phagocytosis mediated by FcR, complement ***receptor*** -mediated phagocytosis did not alter H2O2 production or arachidonic acid release. Ligation of FcR was not sufficient to depress triggered H2O2 production and arachidonic acid release because these functions were not depressed when phagocytosis was inhibited with cytochalasin D. Thus, it was found that the depression of triggered H2O2 production by macrophages after FcR-mediated phagocytosis was associated with impaired release of arachidonic acid and that H2O2 production could be partially restored by the addition of arachidonic acid. These results suggest that the impairment of arachidonic acid release after FcR-mediated phagocytosis contributes to the depression of macrophage respiratory burst capacity after FcR-mediated phagocytosis.

TI DEPRESSION OF MACROPHAGE RESPIRATORY BURST CAPACITY AND ARACHIDONIC-ACID RELEASE AFTER FC ***RECEPTOR*** -MEDIATED PHAGOCYTOSIS

AB . . . phagocytosis of IgG-coated erythrocytes (EIgG) by macrophages results in a subsequent depression of macrophage phagocytic function, respiratory burst capacity, and ***bactericidal*** activity. Our study was carried out to determine the importance of impaired arachidonic acid release in the depression of the . . . exogenous arachidonic acid partially prevented the depression of triggered H2O2 production after EIgG phagocytosis. Unlike phagocytosis mediated by FcR, complement ***receptor*** -mediated phagocytosis did not alter H2O2 production or arachidonic acid release. Ligation of FcR was not sufficient to depress triggered H2O2. . .

STP KeyWords Plus (R): HYDROGEN-PEROXIDE PRODUCTION; NEUTROPHIL NADPH-OXIDASE; UNSATURATED FATTY-ACIDS; SUPEROXIDE GENERATION; ERYTHROCYTE- ***GHOSTS*** ; PROTEIN-SYNTHESIS; HOST DEFENSE; ACTIVATION; OXYGEN; INHIBITORS

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AN 1993:165142 BIOSIS <<LOGINID::20091202>>

DN PREV199395086192

TI Fc-epsilon-RI-mediated hydrolysis of phosphoinositides in permeable membrane vesicles.

AU Kuhn, Donald E.; Dreskin, Stephen C. [Reprint author]

CS B164, Univ. Colo. Sch. Med., 4200 East Ninth Ave., Denver, CO 80262, USA

SO Journal of Immunological Methods, (1993) Vol. 157, No. 1-2, pp. 81-89. CODEN: JIMMBG. ISSN: 0022-1759.

DT Article

LA English

ED Entered STN: 31 Mar 1993

Last Updated on STN: 1 Apr 1993

AB We have used hypotonic lysis of cytoplasm derived from rat basophilic

leukemia (RBL) cell to prepare organelle and cytoplasm-depleted membrane vesicles called 'RBL cell ***ghosts***' (Dreskin and Metzger, 1991). Unlike other membrane preparations, the RBL ***ghosts*** hydrolyze phosphoinositides (PIs) in response to aggregation of the high affinity ***receptor*** for IgE (Fc-epsilon-RI) and have proven useful for studies of the molecular events involved in transduction of this signal. A significant limitation of these preparations is that they are sealed. Thus, to incorporate membrane-impermeant molecules (such as ATP) into the intravesicular space of the ***ghosts***, they must be added as the ***ghosts*** are formed. We have now overcome this problem by permeabilizing the ***ghosts*** with alpha-toxin from *S. aureus* and find that, following permeabilization, ***ghosts*** activated via Fc-epsilon-RI, hydrolyze PIs for a longer time than do non-permeabilized ***ghosts***. As in the intact ***ghosts***, this response is absolutely dependent upon ATP and is enhanced by the addition of either phosphoenolpyruvate (PEP) or creatine phosphate (CP). This report demonstrates that we can now manipulate the intravesicular environment of the RBL ***ghosts*** and extends the utility of these preparations as a model system for the study of signal transduction following activation via Fc-epsilon-RI.

AB. . . lysis of cytoplasm derived from rat basophilic leukemia (RBL) cell to prepare organelle and cytoplasm-depleted membrane vesicles called 'RBL cell ***ghosts***' (Dreskin and Metzger, 1991). Unlike other membrane preparations, the RBL ***ghosts*** hydrolyze phosphoinositides (PIs) in response to aggregation of the high affinity ***receptor*** for IgE (Fc-epsilon-RI) and have proven useful for studies of the molecular events involved in transduction of this signal. A. . . preparations is that they are sealed. Thus, to incorporate membrane-impermeant molecules (such as ATP) into the intravesicular space of the ***ghosts***, they must be added as the ***ghosts*** are formed. We have now overcome this problem by permeabilizing the ***ghosts*** with alpha-toxin from *S. aureus* and find that, following permeabilization, ***ghosts*** activated via Fc-epsilon-RI, hydrolyze PIs for a longer time than do non-permeabilized ***ghosts***. As in the intact ***ghosts***, this response is absolutely dependent upon ATP and is enhanced by the addition of either phosphoenolpyruvate (PEP) or creatine phosphate (CP). This report demonstrates that we can now manipulate the intravesicular environment of the RBL ***ghosts*** and extends the utility of these preparations as a model system for the study of signal transduction following activation via. . .

IT Miscellaneous Descriptors

ALPHA TOXIN PERMEABILIZING AGENT; ATP DEPENDENT; CREATINE PHOSPHATE; HIGH AFFINITY IMMUNOGLOBULIN E ***RECEPTOR***; HIGH ENERGY PHOSPHATE COMPOUND ENHANCEMENT; METHOD; PHOSPHOENOLPYRUVATE; RAT BASOPHILIC LEUKEMIA CELL ***GHOSTS***; SIGNAL TRANSDUCTION

ORGN Classifier

Micrococcaceae 07702

Super Taxa

Gram-Positive Cocci; Eubacteria; ***Bacteria***; Microorganisms

Organism Name

Staphylococcus aureus

Taxa Notes

Bacteria, Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name
Muridae
Taxa Notes
Animals, Chordates, . . .

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STN DUPLICATE 7

AN 1992:456099 BIOSIS <<LOGINID::20091202>>
DN PREV199294097499; BA94:97499

TI SURVEY OF A ***RECEPTOR*** PROTEIN IN HUMAN ERYTHROCYTES FOR
HEMAGGLUTININ OF PORPHYROMONAS-GINGIVALIS.

AU HAYASHI H [Reprint author]; NAGATA A; HINODE D; SATO M; NAKAMURA R
CS DEP OF PREVENTIVE DENTISTRY, UNIV OF TOKUSHIMA SCH OF DENT, 18-15,
KURAMOTOCHO-3-CHROME, TOKUSHIMA CITY 770, JAPAN
SO Oral Microbiology and Immunology, (1992) Vol. 7, No. 4, pp. 204-211.
CODEN: OMIMEE. ISSN: 0902-0055.

DT Article
FS BA
LA ENGLISH
ED Entered STN: 7 Oct 1992
Last Updated on STN: 7 Oct 1992

AB The purpose of this study is to survey a ***receptor*** protein in
human erythrocyte membrane for the hemagglutinin (HA) of Porphyromonas
gingivalis. Human erythrocytes were modified by either chymotrypsin or P.
gingivalis HA along with the disappearance of their hemagglutinating
ability and the removal of the band 3 protein. By preparative
electrophoresis, this protein was isolated and purified from human
erythrocytes. The purified protein showed strong inhibitory activity for
hemagglutination and the binding to P. gingivalis cells, whose binding
sites were calculated to be approximately 9000, suggesting its binding to
the active site of HA. Hemagglutinin purified from P. gingivalis by
affinity absorption to sheep erythrocyte ***ghosts*** possessed strong
trypsin-like activity, and both the HA and the enzyme activities were
inhibited by arginine. Specific modification of arginyl residues in human
erythrocytes by phenylglyoxal diminished the hemagglutinating ability.
From the similarity of the inhibition profile and possible active sites
between HA and the trypsin-like protease, it is suggested that
hemagglutination may occur as a result of the primary reaction of the
enzyme (protease) and the substrate. These results suggest that band 3
may be a key protein in human erythrocyte membrane for HA from P.
gingivalis and its binding sites may be arginyl residues of the protein.

TI SURVEY OF A ***RECEPTOR*** PROTEIN IN HUMAN ERYTHROCYTES FOR
HEMAGGLUTININ OF PORPHYROMONAS-GINGIVALIS.

AB The purpose of this study is to survey a ***receptor*** protein in
human erythrocyte membrane for the hemagglutinin (HA) of Porphyromonas
gingivalis. Human erythrocytes were modified by either chymotrypsin or.
. . suggesting its binding to the active site of HA. Hemagglutinin
purified from P. gingivalis by affinity absorption to sheep erythrocyte
ghosts possessed strong trypsin-like activity, and both the HA
and
the enzyme activities were inhibited by arginine. Specific modification
of arginyl. . .

ORGN Classifier
Bacteroidaceae 06901
Super Taxa
Anaerobic Gram-Negative Rods; Eubacteria; ***Bacteria*** ;
Microorganisms

Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, . . .

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 STN DUPLICATE 8

AN 1992:236269 BIOSIS <<LOGINID::20091202>>
 DN PREV199293124294; BA93:124294
 TI SOLUBILIZATION OF THE BINDING PROTEIN FROM EHRLICH ASCITES CELLS AND
 ERYTHROCYTES TO PSEUDOMONAS-AERUGINOSA CYTOTOXIN.
 AU JUNGBLUT R [Reprint author]; GRIMMIG M; LEIDOLF R; LUTZ F
 CS INST PHARMAKOLOGIE TOXIKOLOGIE, JUSTUS-LIEBIG-UNIVERSITAET GIESSEN,
 FRANKFURTER STR 107, W-6300 GIESSEN, GERMANY
 SO Biological Chemistry Hoppe-Seyler, (1992) Vol. 373, No. 2, pp. 93-100.
 CODEN: BCHSEI. ISSN: 0177-3593.
 DT Article
 FS BA
 LA ENGLISH
 ED Entered STN: 10 May 1992
 Last Updated on STN: 10 May 1992

AB The binding protein for pore-forming Pseudomonas aeruginosa cytotoxin was
 solubilized from Ehrlich ascites cell plasma membranes and rabbit and
 bovine erythrocyte ***ghosts*** using nonionic and zwittergent
 detergents. Analysis of solubilized plasma membranes from Ehrlich cells
 by a ***ligand*** -blot technique after separation by
 SDS-PAGE/electrophoretic transfer to nitrocellulose or affinity
 chromatography showed a protein of 70 kDa molecular mass, which binds to
 cytotoxin. The binding protein solubilized from rabbit erythrocyte
 ghosts showed a molecular mass of 50 kDa and that from bovine
 ghosts 55 kDa according to the former test. The binding proteins
 could be characterized as acidic. They contain a glycan moiety which is,
 however, not involved in the interaction of cytotoxin with the binding
 site.

AB. . . binding protein for pore-forming Pseudomonas aeruginosa cytotoxin
 was solubilized from Ehrlich ascites cell plasma membranes and rabbit and
 bovine erythrocyte ***ghosts*** using nonionic and zwittergent
 detergents. Analysis of solubilized plasma membranes from Ehrlich cells
 by a ***ligand*** -blot technique after separation by
 SDS-PAGE/electrophoretic transfer to nitrocellulose or affinity
 chromatography showed a protein of 70 kDa molecular mass, which binds to
 cytotoxin. The binding protein solubilized from rabbit erythrocyte
 ghosts showed a molecular mass of 50 kDa and that from bovine
 ghosts 55 kDa according to the former test. The binding proteins
 could be characterized as acidic. They contain a glycan moiety. . .

ORGN Classifier
 Pseudomonadaceae 06508
 Super Taxa
 Gram-Negative Aerobic Rods and Cocci; Eubacteria; ***Bacteria*** ;
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Bovidae 85715
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman. . .

L7 ANSWER 36 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 1992:1761 CAPLUS <<LOGINID::20091202>>

DN 116:1761

OREF 116:363a,366a

TI Membrane-anchoring of heterologous proteins in recombinant hosts for use as antigens

IN Lubitz, Werner; Szostak, Michael P.

PA Boehringer Mannheim G.m.b.H., Germany

SO PCT Int. Appl., 46 pp.

CODEN: PIXXD2

DT Patent

LA German

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	----	-----	-----	-----
PI	WO 9113155	A1	19910905	WO 1991-EP308	19910219
	W: AU, FI, HU, JP, SU, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
	DE 4005874	A1	19911107	DE 1990-4005874	19900224
	AU 9172373	A	19910918	AU 1991-72373	19910219
	EP 516655	A1	19921209	EP 1991-903789	19910219
	EP 516655	B1	19940504		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 05503014	T	19930527	JP 1991-503980	19910219
	JP 3238396	B2	20011210		
	AT 105335	T	19940515	AT 1991-903789	19910219
	US 5470573	A	19951128	US 1992-924028	19920930
PRAI	DE 1990-4005874	A	19900224		
	EP 1991-903789	A	19910219		
	WO 1991-EP308	A	19910219		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Antigenic proteins are prepd. with a Gram-neg. ***bacteria*** contg. a gene encoding a lytic protein by expression of a chimeric gene for a fusion protein of a membrane-anchoring domain and the antigen. Plasmid pAV5 encoding a streptavidin-phage MS2 protein L fusion protein and a plasmid contg. the protein E gene of phage .phi.X174 under control of the temp. sensitive .lambda. repressor-.lambda. promoter/operator system were prepd. Escherichia coli was transformed with these plasmids, cultured to permit cell growth and fusion protein synthesis, then temp.-shifted to cause protein E prodn. and cell lysis. The ***bacterial***
ghosts prepd. were incubated with a hepatitis B core antigen-
biotin conjugate to prep. an immunogen.

OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Antigenic proteins are prepd. with a Gram-neg. ***bacteria*** contg. a gene encoding a lytic protein by expression of a chimeric gene for a fusion protein of a membrane-anchoring. . . cultured to permit cell growth and fusion protein synthesis, then temp.-shifted to cause protein E prodn. and cell lysis. The ***bacterial*** ***ghosts*** prepd. were incubated with a hepatitis B core antigen- ***biotin*** conjugate

to prep. an immunogen.

ST antigen membrane anchor fusion Escherichia; lytic protein
 bacterial ***ghost*** immunogen; vaccine recombinant
 bacteria ***ghost***

IT Vaccines
 (***bacterial*** ***ghosts*** contg. membrane-assocd.
 recombinant antigens for, prepn. of)

IT Avidins
 RL: PREP (Preparation)
 (fusion products with membrane-anchoring domains, recombinant manuf. in
 Escherichia coli of, prepn. of cell ***ghosts*** for vaccines of,
 bacteriophage lytic functions in)

IT Antigens
 RL: PREP (Preparation)
 (fusion proteins with membrane-anchoring domains of, Gram-neg.
 bacterial ***ghosts*** contg., prepn. of,
 bacteriophage lytic functions in, vaccines in relation to)

IT Escherichia coli
 (***ghosts*** of, antigens anchored to membranes of,
 bacteriophage lytic functions in, vaccines in relation to)

IT Virus, ***bacterial***
 (lytic functions of, in prepn. Gram-neg. ***bacterial***
 ghosts contg. antigen-membrane-anchoring domain fusion
 proteins, vaccines in relation to)

IT Proteins, biological studies
 RL: PREP (Preparation)
 (lytic, of ***bacteriophage*** , in prepn. Gram-neg.
 bacterial ***ghosts*** contg. of antigen-membrane-
 anchoring
 domain fusion proteins, vaccines in relation to)

IT Mammal
 (vaccines for, antigens for, ***bacterial*** ***ghosts***
 contg. membrane-assocd. recombinant antigens as)

IT Proteins, specific or class
 RL: PREP (Preparation)
 (E, of ***bacteriophage*** .phi.X174, in prepn. of Gram-neg.
 bacterial ***ghosts*** contg. antigen-membrane-anchoring
 domain fusion proteins, vaccines in relation to)

IT Proteins, specific or class
 RL: PREP (Preparation)
 (L, of ***bacteriophage*** MS2, in prepn. of Gram-neg.
 bacterial ***ghosts*** contg. antigen-membrane-anchoring
 domain fusion proteins, vaccines in relation to)

IT Virus, ***bacterial***
 (MS2, protein L of, in prepn. Gram-neg. ***bacterial***
 ghosts contg. antigen-membrane-anchoring domain fusion
 proteins, vaccines in relation to)

IT Gene
 RL: BIOL (Biological study)
 (chimeric, for fusion proteins of antigens and membrane-anchoring
 domains, expression in Gram-neg. ***bacteria*** of, vaccines in
 relation to)

IT Proteins, specific or class
 RL: BIOL (Biological study)
 (fusion products, of antigens with membrane-anchoring domain, manuf. in
 Gram-neg. ***bacteria*** of, ***bacteriophage*** lytic
 functions in, vaccine manuf. in relation to)

IT Sialoglycoproteins
 RL: PREP (Preparation)
 (gp120env, fusion products, with ***bacteriophage*** proteins E or L, membrane anchoring in Escherichia coli of, prepn. of ***ghosts*** for vaccines in relation to)

IT Glycoproteins, specific or class
 RL: PREP (Preparation)
 (gp41env, fusion products, with ***bacteriophage*** proteins E or L, membrane anchoring in Escherichia coli of, prepn. of ***ghosts*** for vaccines in relation to)

IT ***Bacteria***
 (gram-neg., membrane anchoring of heterologous proteins in, membrane-anchoring domains and ***bacteriophage*** lytic functions in, vaccines in relation to)

IT Antigens
 RL: BIOL (Biological study)
 (hepatitis B core, conjugate with ***biotin*** , complex with Escherichia coli ***ghosts*** contg. membrane-bound streptavidin, as immunogen)

IT Virus, ***bacterial***
 (phi X174, protein E of, in prepn. Gram-neg. ***bacterial*** ***ghosts*** contg. antigen-membrane-anchoring domain fusion proteins, vaccines in relation to)

IT 137925-62-3, Deoxyribonucleic acid (Escherichia coli clone pMC1403 gene lacZ plus 3'-flanking region fragment) 137925-65-6 137926-10-4, Deoxyribonucleic acid (Streptomyces avidinii clone pAV5 streptavidin gene plus 5'- and 3'-flanking region fragment)
 RL: BIOL (Biological study)
 (chimeric gene contg., for fusion protein of membrane-anchoring domain and antigenic determinant, expression in Escherichia coli of, ***bacteriophage*** lytic functions in)

IT 9013-20-1D, Streptavidin, fusion products with membrane-anchoring protein 9031-11-2D, .beta.-Galactosidase, fusion products with phage E or L proteins
 RL: BIOL (Biological study)
 (membrane-bound, recombinant manuf. in Escherichia coli of, prepn. of cell ***ghosts*** for vaccines of, ***bacteriophage*** lytic functions in)

L7 ANSWER 37 OF 68 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

AN 1992046723 EMBASE <<LOGINID::20091202>>

TI Comparison of hemagglutinating pili of Haemophilus influenzae type b with similar structures of nontypeable H. influenzae.

AU Gilsdorf, J.R. (correspondence); Chang, H.Y.; McCrea, K.W.; Bakaletz, L.O.

CS Department of Pediatrics, University of Michigan, Ann Arbor, MI 48109-0244, United States.

SO Infection and Immunity, (1991) Vol. 60, No. 2, pp. 374-379.
 ISSN: 0019-9567 CODEN: INFIBR

CY United States

DT Journal; Article

FS 004 Microbiology: Bacteriology, Mycology, Parasitology and Virology

LA English

SL English

ED Entered STN: 20 Mar 1992
 Last Updated on STN: 20 Mar 1992

AB Thirty-eight clinical isolates of nontypeable Haemophilus influenzae were

tested for the presence of hemagglutinating pili similar to those of H. influenzae type b (Hib) that mediate buccal epithelial cell adherence. Four endogenously hemagglutinating (HA+) strains were identified, and eight additional HA+ variants were obtained from HA- strains by erythrocyte enrichment. All 12 HA+ nontypeable H. influenzae isolates bound antisera directed against denatured pilins of Hib, but none bound antisera against assembled native pili of Hib. In erythrocyte- and buccal-cell-binding assays, HA+ nontypeable H. influenzae binding was reduced compared with HA+ Hib binding and was not significantly different from HA- nontypeable H. influenzae binding. Both HA- and HA+ nontypeable H. influenzae binding was increased over binding of HA- Hib. HA+ nontypeable H. influenzae strains agglutinated adult erythrocytes that possess the Anton antigen, which is thought to be the ***receptor*** for Hib pili, and did not agglutinate cord or Lu(a-b-) dominant erythrocytes, which lack the Anton antigen. Electron microscopy of HA- and HA+ variants of three nontypeable H. influenzae strains showed few or no surface appendages on the HA- organisms, but piluslike structures were seen on many organisms from two HA+ nontypeable H. influenzae strains and on a few organisms from one strain. Thus, nontypeable H. influenzae appears to possess structures that are immunologically similar to the pilins that make up the hemagglutinating pili of Hib. However, nontypeable H. influenzae appears to also possess mechanisms for erythrocyte and buccal cell adherence that are not directly correlated with the presence of a hemagglutinating pilus.

AB . . . Hib. HA+ nontypeable H. influenzae strains agglutinated adult erythrocytes that possess the Anton antigen, which is thought to be the ***receptor*** for Hib pili, and did not agglutinate cord or Lu(a-b-) dominant erythrocytes, which lack the Anton antigen. Electron microscopy of. . .

CT Medical Descriptors:
article

****bacterium adherence***
****bacterium identification***
****bacterium pilus***
cheek mucosa
comparative study
controlled study
electron microscopy
epithelium cell
erythrocyte ghost
*haemophilus influenzae type b
*hemagglutination
human
human cell
pasteurellaceae
phenotype
priority journal
*polyclonal antibody
*rabbit antiserum

L7 ANSWER 38 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1991:70460 BIOSIS <<LOGINID::20091202>>

DN PREV199191039120; BA91:39120

TI INHIBITION OF CHOLERA TOXIN BINDING TO MEMBRANE RECEPTORS BY PIG GASTRIC MUCIN-DERIVED GLYCOPEPTIDES DIFFERENTIAL EFFECT DEPENDING ON THE ABO BLOOD GROUP ANTIGENIC DETERMINANTS.

AU MONFERRAN C G [Reprint author]; ROTH G A; CUMAR F A
CS DEP DE QUIMICA BIOL, FAC DE CIENCIAS QUIMICAS, UNIV NACIONAL DE CORDOBA,
CIQUIBIC-CONICET, 5016 CORDOBA, ARGENTINA
SO Infection and Immunity, (1990) Vol. 58, No. 12, pp. 3966-3972.
CODEN: INFIBR. ISSN: 0019-9567.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 29 Jan 1991
Last Updated on STN: 30 Jan 1991
AB The capacity of pig gastric mucin-derived glycopeptides to interfere with
the binding of cholera toxin (CT) to membrane receptors was studied. Two
types of glycopeptide preparations with or without human blood group A
antigenic activity were assayed for comparison in a system in which the
target for the toxin was rat erythrocyte ***ghosts***. Blood group
A-active glycopeptides (A+ glycopeptides) were more potent inhibitors for
the toxin binding than those lacking group A activity (A- glycopeptides).
The mean values of the 50% inhibitory dose revealed that the A+
glycopeptide preparations were 6.6-fold-more potent inhibitors than the A-
ones (P < 0.001). The inhibitory capacity of the different A+
glycopeptide preparations was not directly proportional to the group A
antigenic titer. The A+ glycopeptides showed a higher capacity than the
A- glycopeptides to interact with the toxin as revealed by CT-glycopeptide
complex formation, which could be detected by Sephacryl S-400
chromatography. This result suggests that glycopeptide inhibition of CT
binding to the erythrocyte ***ghosts*** is mediated by a competition
between the GM1 receptors and the glycopeptides for the toxin. The
differential effect between both types of glycoconjugates was independent
of the way of measuring the amount of glycopeptides used (dry weight,
carbohydrate or protein content). The existence in the gastrointestinal
tract of mucins not carrying or carrying different ABO blood group
determinants, which could behave as more or less potent inhibitors of CT
binding to membrane receptors, may help to explain the relationship
between ABO blood groups and severity of cholera.
AB. . . A antigenic activity were assayed for comparison in a system in
which the target for the toxin was rat erythrocyte ***ghosts***.
Blood group A-active glycopeptides (A+ glycopeptides) were more potent
inhibitors for the toxin binding than those lacking group A activity. .
. which could be detected by Sephacryl S-400 chromatography. This result
suggests that glycopeptide inhibition of CT binding to the erythrocyte
ghosts is mediated by a competition between the GM1 receptors and
the glycopeptides for the toxin. The differential effect between both. .
.
IT Miscellaneous Descriptors
ANTIGENIC TITER GLYCOCONJUGATE TYPES ***RECEPTOR*** COMPETITION
ORGN Classifier
Vibrionaceae 06704
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms
ORGN Classifier
Suidae 85740
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes

Animals, Artiodactyls, Chordates, Mammals, Nonhuman. . .

L7 ANSWER 39 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 9

AN 1991:114458 BIOSIS <<LOGINID::20091202>>

DN PREV199191061848; BA91:61848

TI EFFECT OF PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE ***GHOSTS*** ON
MACROPHAGE PHAGOCYTIC FUNCTION AND HYDROGEN PEROXIDE PRODUCTION.

AU COMMINS L M [Reprint author]; LOEGERING D J; GUDEWICZ P W

CS DEP PHYSIOLOGY CELL BIOL, ALBANY MED COLL, 47 NEW SCOTLAND AVENUE, ALBANY,
NEW YORK 12208, USA

SO Inflammation, (1990) Vol. 14, No. 6, pp. 705-716.
CODEN: INFLD4. ISSN: 0360-3997.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 27 Feb 1991
Last Updated on STN: 28 Feb 1991

AB Our previous studies have shown that an in vivo phagocytic challenge with
IgG-coated erythrocytes can depress Kupffer cell complement and Fc
receptor function, as well as decrease the survival rate
following
endotoxemia and ***bacteremia***. In an effort to better understand
the mechanism underlying these in vivo findings, the present study
evaluated the in vitro effects of a phagocytic challenge with either
IgG-coated erythrocytes (EIgG) or erythrocyte ***ghosts*** (GIgG) on
macrophage phagocytic and respiratory burst activity. Elicited rat
peritoneal macrophage (PM) monolayers were challenged with varying doses
of EIgG, then the noninternalized EIgG were lysed hypotonically and the
monolayers incubated for an additional hour prior to determining
phagocytic function and PMA-stimulated hydrogen peroxide production.
Challenge of PM with 1 .times. 106 EIgG per well had no effect, but
challenge with 1 .times. 107 or 1 .times. 108 EIgG per well caused a
dose-dependent depression of phagocytic function or hydrogen peroxide
production. GIgG were formed by hypotonically lysing EIgG bound to PM at
4.degree. C. The bound GIgG were phagocytized during a subsequent
incubation at 37.degree. C. Challenge with GIgG depressed phagocytic
function only with the highest challenge dose tested (1 .times. 108 per
well) and did not depress hydrogen peroxide production. The observation
that prior phagocytic challenge with EIgG depressed macrophage function to
a greater extent than challenge with GIgG supports our previous in vivo
observations. Furthermore, these studies suggest that the internalization
of erythrocyte contents, and not phagocytosis per se, plays an important
role in determining macrophage host defense function.

TI EFFECT OF PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE ***GHOSTS*** ON
MACROPHAGE PHAGOCYTIC FUNCTION AND HYDROGEN PEROXIDE PRODUCTION.

AB. . . previous studies have shown that an in vivo phagocytic challenge
with IgG-coated erythrocytes can depress Kupffer cell complement and Fc
receptor function, as well as decrease the survival rate
following
endotoxemia and ***bacteremia***. In an effort to better understand
the mechanism underlying these in vivo findings, the present study
evaluated the in vitro effects of a phagocytic challenge with either
IgG-coated erythrocytes (EIgG) or erythrocyte ***ghosts*** (GIgG) on
macrophage phagocytic and respiratory burst activity. Elicited rat
peritoneal macrophage (PM) monolayers were challenged with varying doses
of. . .

L7 ANSWER 40 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
 AN 1990:26846 BIOSIS <<LOGINID::20091202>>
 DN PREV199089013812; BA89:13812
 TI NEURAMINIDASE OF NONPATHOGENIC ARTHROBACTER-SP.
 AU KOTLYAR T V [Reprint author]; SHATAEVA L K; ZAIKINA N A; CHERNOVA I A; ABRASHEV I R
 CS LENINGR CHEM PHARM INST, LENINGRAD, USSR
 SO Prikladnaya Biokhimiya i Mikrobiologiya, (1989) Vol. 25, No. 4, pp. 467-472.
 CODEN: PBMIAK. ISSN: 0555-1099.
 DT Article
 FS BA
 LA RUSSIAN
 ED Entered STN: 19 Dec 1989
 Last Updated on STN: 20 Dec 1989
 AB Neuraminidase of the nonpathogenic microorganism *Arthrobacter nicotianae* was studied by gel chromatography and affinity chromatography on "Stropan"-an adsorbent containing erythrocyte ***ghosts*** incorporated in a porous polymeric matrix. The molecular weight of the enzyme was found to be 170 .+- . 20 kDa. The conditions of the neuraminidase adsorption on Stropan were optimized. The neuraminidase preparation obtained is comparable with commercial neuraminidase from *noncholerae vibri*one by the specific activity and the specific effect on receptors of human erythrocytes.
 AB. . . Neuraminidase of the nonpathogenic microorganism *Arthrobacter nicotianae* was studied by gel chromatography and affinity chromatography on "Stropan"-an adsorbent containing erythrocyte ***ghosts*** incorporated in a porous polymeric matrix. The molecular weight of the enzyme was found to be 170 .+- . 20 kDa.. . .
 IT Miscellaneous Descriptors
 HUMAN ERYTHROCYTE ***RECEPTOR*** OPTIMIZATION AFFINITY
 CHROMATOGRAPHY BIOTECHNOLOGY INDUSTRY
 ORGN Classifier
 Irregular Nonsporing Gram-Positive Rods 08890
 Super Taxa
 Actinomycetes and Related Organisms; Eubacteria; ***Bacteria*** ;
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms
 ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates,. . .
 L7 ANSWER 41 OF 68 LIFESCI COPYRIGHT 2009 CSA on STN
 AN 89:169 LIFESCI <<LOGINID::20091202>>
 TI Novel lectins derived from ***bacterial*** pili.
 AU Brinton, C.C., Jr.; Hanson, M.
 CS Bactex, Inc., Pittsburgh, PA (USA)
 PI US 4801690 1989
 SO (1989) . US Cl. 530/396; Int. Cl. A61K 37/02, 39/108, 39/112, C12N 1/00..
 DT Patent
 FS A; W

LA English

AB The authors describe a bactolectin derived from the pili of an organism selected from the group consisting of E. coli , and Salmonella species, said ***lectin*** being non-covalently bindable to the pilus rod protein of said pili and separable therefrom by the action of hot aqueous sodium dodecyl sulfate and possessing at least a single binding site for binding to mammalian erythrocyte ***ghosts*** .

TI Novel lectins derived from ***bacterial*** pili.

AB . . . derived from the pili of an organism selected from the group consisting of E. coli , and Salmonella species, said ***lectin*** being non-covalently bindable to the pilus rod protein of said pili and separable therefrom by the action of hot aqueous sodium dodecyl sulfate and possessing at least a single binding site for binding to mammalian erythrocyte ***ghosts*** .

UT patents; lectins; Escherichia coli; Salmonella; pili; ***bacteria***

L7 ANSWER 42 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN

AN 1988:183579 CAPLUS <<LOGINID::20091202>>

DN 108:183579

OREF 108:30088h,30089a

TI Purifications of lectins containing mammalian erythrocyte binding sites from ***bacterial*** pili

IN Brinton, Charles C., Jr.; Hanson, Mark

PA Bactex, Inc., USA

SO PCT Int. Appl., 27 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----	----	-----	-----	-----
PI	WO 8705910	A1	19871008	WO 1987-US617	19870320
	W: JP				
	RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
	US 4801690	A	19890131	US 1986-842946	19860324
	EP 298991	A1	19890118	EP 1987-902927	19870320
	EP 298991	B1	19920617		
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	JP 01501225	T	19890427	JP 1987-502341	19870320
	JP 2778690	B2	19980723		
	AT 77392	T	19920715	AT 1987-902927	19870320
	CA 1282323	C	19910402	CA 1987-532748	19870323
PRAI	US 1986-842946	A	19860324		
	US 1986-842947	A	19860324		
	EP 1987-902927	A	19870320		
	WO 1987-US617	W	19870320		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB ***Lectin*** is isolated from pili of pilated ***bacteria*** .

The lectins are noncovalently bindable to the pilus rod protein of the pili and possess a single binding site for mammalian erythrocyte ***ghosts*** . Escherichia coli Type I was suspended in aq. NaCl and blended. The product was centrifuged to remove cell debris. The supernatant was taken in buffer contg. SDS, boiled for 5 min, cooled, and aggregated pilin rods were sedimented by centrifugation. The supernatant contained 28-, 16.5-, and 14.5-kilodalton proteins, which were pptd. with acetone and sepd. by Sephadex gel column. Papain inactivation of pili was correlated with degrdn. of the 28-kilodalton protein on the pili. This

lectin was coupled to CNBr Sepharose for use as mannose-specific affinity resin.

OSC.G 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Purifications of lectins containing mammalian erythrocyte binding sites from ***bacterial*** pili

AB ***Lectin*** is isolated from pili of piliated ***bacteria*** . The lectins are noncovalently bindable to the pilus rod protein of the pili and possess a single binding site for mammalian erythrocyte ***ghosts*** . Escherichia coli Type I was suspended in aq. NaCl and blended. The product was centrifuged to remove cell debris. The . . . by Sephadex gel column. Papain inactivation of pili was correlated with degrdn. of the 28-kilodalton protein on the pili. This ***lectin*** was coupled to CNBr Sepharose for use as mannose-specific affinity resin.

ST ***lectin*** purifn pili ***bacteria*** ; Escherichia pili
lectin purifn

IT Pili
(***lectin*** purifn. from, of ***bacteria*** , erythrocyte binding site of)

IT Escherichia coli
Klebsiella pneumoniae
Neisseria gonorrhoeae
Neisseria meningitidis
Pseudomonas aeruginosa
Salmonella
Streptococcus pneumoniae
(pili of, ***lectin*** contg. erythrocyte binding site purifn. from)

IT Agglutinins and Lectins
RL: BIOL (Biological study)
(purifn. of erythrocyte binding site-contg., of ***bacterial*** pili)

IT Erythrocyte
(***ghost*** , binding site for, on ***lectin*** of ***bacterial*** pili)

IT 3458-28-4, Mannose
RL: BIOL (Biological study)
(binding to, ***lectin*** of Escherichia coli pili specific for)

IT 9001-73-4, Papain
RL: BIOL (Biological study)
(erythrocyte binding site of ***bacterial*** pili ***lectin*** inactivation with)

IT 137-16-6 151-21-3, SDS, biological studies
RL: BIOL (Biological study)
(in ***lectin*** purifn. from ***bacterial*** pili, erythrocyte binding site in relation to)

IT 9012-36-6DP, Sepharose, cyanobromide deriv., reaction products with ***lectin*** of Escherichia pili
RL: PREP (Preparation)
(prepn. of, as mannose-specific affinity probe)

L7 ANSWER 43 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN DUPLICATE 10

AN 1987:463133 BIOSIS <<LOGINID::20091202>>

DN PREV198784108573; BA84:108573

TI EFFECT OF KUPFFER CELL PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE

GHOSTS ON SUSCEPTIBILITY TO ENDOTOXEMIA AND ***BACTEREMIA*** .
 AU LOEGERING D J [Reprint author]; COMMINES L M; MINNEAR F L; GARY L A; HILL L A
 CS DEP PHYSIOLOGY, NEIL HELLMAN MED RES BUILD, ALBANY MED COLL UNION UNIV, ALBANY, NY 12208, USA
 SO Infection and Immunity, (1987) Vol. 55, No. 9, pp. 2074-2080. CODEN: INFIBR. ISSN: 0019-9567.
 DT Article
 FS BA
 LA ENGLISH
 ED Entered STN: 7 Nov 1987
 Last Updated on STN: 7 Nov 1987
 AB The phagocytosis of erythrocytes by macrophages has previously been shown to depress macrophage function. In this study we compared the effect of the phagocytosis of erythrocytes and erythrocyte ***ghosts*** by Kupffer cells on the duration of the depression of complement ***receptor*** clearance function and host defense against endotoxemia and ***bacteremia*** . Phagocytosis of erythrocytes and erythrocyte ***ghosts*** was induced in rats by the injection of rat erythrocytes or erythrocyte ***ghosts*** coated with anti-rat erythrocyte immunoglobulin G (EIgG and GIgG, respectively). The hepatic uptake of EIgG and GIgG (17.4 .times. 108/100 g) occurred during the first 30 min after injection. The digestion of phagocytized EIgG and GIgG, as assessed by electron microscopy, was complete at 24 and 3 h after injection, respectively. The depression of Kupffer cell complement ***receptor*** clearance function caused by EIgG and GIgG returned to normal by 6 h after injection of EIgG and by 3 h after injection of GIgG. Phagocytosis of EIgG depressed the survival rate after endotoxemia and ***bacteremia*** when endotoxin or ***bacteria*** were injected at 30 min after EIgG. The survival rate returned to normal when the endotoxin and ***bacteria*** were injected at 12 and 6 h after the EIgG, respectively.
 Phagocytosis of GIgG did not depress the survival rate after endotoxemia and ***bacteremia*** . Thus, compared with erythrocytes, erythrocyte ***ghosts*** are more rapidly digested after phagocytosis, depress complement ***receptor*** function for a shorter period of time, and cause less depression of host defense. These findings indicate that the contents of erythrocytes play an important role in the impairment of host defense caused by the phagocytosis of erythrocytes by Kupffer cells.
 TI EFFECT OF KUPFFER CELL PHAGOCYTOSIS OF ERYTHROCYTES AND ERYTHROCYTE ***GHOSTS*** ON SUSCEPTIBILITY TO ENDOTOXEMIA AND ***BACTEREMIA*** .
 AB. . . . been shown to depress macrophage function. In this study we compared the effect of the phagocytosis of erythrocytes and erythrocyte ***ghosts*** by Kupffer cells on the duration of the depression of complement ***receptor*** clearance function and host defense against endotoxemia and ***bacteremia*** . Phagocytosis of erythrocytes and erythrocyte ***ghosts*** was induced in rats by the injection of rat erythrocytes or erythrocyte ***ghosts*** coated with anti-rat erythrocyte immunoglobulin G (EIgG and GIgG, respectively). The hepatic uptake of EIgG and GIgG (17.4 .times. 108/100. . . . assessed by electron microscopy, was complete at 24 and 3 h after injection, respectively. The depression of Kupffer cell complement ***receptor*** clearance function caused by EIgG and GIgG returned to normal by 6 h after injection of EIgG and by 3 h after injection of GIgG. Phagocytosis of EIgG depressed the survival rate after endotoxemia and ***bacteremia*** when endotoxin or ***bacteria*** were injected at 30 min after EIgG.

The survival rate returned to normal when the endotoxin and
bacteria were injected at 12 and 6 h after the EIgG,
respectively.

Phagocytosis of GIgG did not depress the survival rate after endotoxemia
and ***bacteremia***. Thus, compared with erythrocytes, erythrocyte
ghosts are more rapidly digested after phagocytosis, depress
complement ***receptor*** function for a shorter period of time, and
cause less depression of host defense. These findings indicate that the
contents. . .

IT Miscellaneous Descriptors

RAT COMPLEMENT ***RECEPTOR*** CLEARANCE DEPRESSION HOST DEFENSE
IMPAIRMENT

L7 ANSWER 44 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 11

AN 1987:66161 BIOSIS <<LOGINID::20091202>>

DN PREV198783034487; BA83:34487

TI ***LECTIN*** BINDING SITES IN PARAMECIUM-TETRAURELIA CELLS II.
LABELING ANALYSIS PREDOMINANTLY OF NON-SECRETORY COMPONENTS.

AU LUETHE N [Reprint author]; PLATTNER H

CS FAC BIOL, UNIV KONSTANZ, POB 5560, D-7750 KONSTANZ, W GER

SO Histochemistry, (1986) Vol. 85, No. 5, pp. 377-388.

CODEN: HCMYAL. ISSN: 0301-5564.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 24 Jan 1987

Last Updated on STN: 24 Jan 1987

AB All the ***lectin*** -FITC conjugates tested (ConA, RCA II, WGA) bind
to the surface of Paramecium cells. Yet only WGA yields a distinct
fluorescent pattern; it contours the basis of cilia and in some cells it
brilliantly stains a few neighbouring rows of the regular surface fields
in the anteroventral region (a region known to contain extensive fields
of linear aggregates of freeze-fracture particles and to be engaged in
conjugation). Incubation in vivo with WGA-FITC resulted in the selective
labeling of the cytopharyngeal region as well as of the cytoproct. On
Lowicryl K4M sections, WGA-gold probes concomitantly labeled disk-shaped
vesicles that are assumed in the literature to serve as shuttle vesicles
between these two cell regions and, thus, to connect forming and
defecating digesting vacuoles (stages DV I and DV IV). On K4M sections
WGA-Au stains also most other components of the lysosomal system. Also on
K4M sections RCA II-Au labeled the walls of ***bacteria*** contained
in DV I and II type digesting vacuoles (but not lysosomes identified bona
fide by their size and shape and by their frequent vicinity to or
continuity with digesting vacuoles). The WGA data largely support
previous conclusions on the possible functional connection of all these
elements (DV I-IV, smaller lysosomes, disk-shaped vesicles etc.) of the
lysosomal system in Paramecium, as proposed by Allen and his group on the
basis of other lines of evidence. As shown in the accompanying paper,
ConA-FITC stained ***ghosts*** (formed after massive trichocyst
exocytosis) also about into DV-like structures. The different results
obtained with the three lectins tested reflect the complex sorting
machinery contained in the elaborate lysosomal system of a Paramecium
cell. In the cytosol, finally, there occurs a particularly intense
staining with ConA-gold, applied to Lowicryl sections, that probably
represents glycogen-like particles. The same procedure reveals some weak
staining of secretory contents and of nuclear structures.

TI ***LECTIN*** BINDING SITES IN PARAMECIUM-TETRAURELIA CELLS II.
 LABELING ANALYSIS PREDOMINANTLY OF NON-SECRETORY COMPONENTS.
 AB All the ***lectin*** -FITC conjugates tested (ConA, RCA II, WGA) bind
 to the surface of Paramecium cells. Yet only WGA yields a distinct
 fluorescent. . . WGA-Au stains also most other components of the
 lysosomal system. Also on K4M sections RCA II-Au labeled the walls of
 bacteria contained in DV I and II type digesting vacuoles (but
 not
 lysosomes identified bona fide by their size and shape. . . Allen and
 his group on the basis of other lines of evidence. As shown in the
 accompanying paper, ConA-FITC stained ***ghosts*** (formed after
 massive trichocyst exocytosis) also about into DV-like structures. The
 different results obtained with the three lectins tested reflect. . .

L7 ANSWER 45 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 1987:191430 CAPLUS <<LOGINID::20091202>>
 DN 106:191430
 OREF 106:30961a,30964a
 TI Characterization and ***receptor*** binding specificities of the
 X-binding UTI Escherichia coli adhesin AFA-I
 AU Schmidt, M. Alexander; Walz, Waltraud; Schoolnik, Gary K.
 CS Sch. Med., Stanford Univ., Stanford, CA, 94305, USA
 SO FEMS Symposium (1986), 31(Protein-Carbohydr. Interact. Biol. Syst.),
 253-62
 CODEN: FEMSDW; ISSN: 0163-9188
 DT Journal
 LA English
 AB AFA-I, a mannose-resistant, P-independent, X-binding afimbrial E. coli
 adhesion was purified from a recombinant strain and chem., functionally,
 and serol. characterized. AFA-I exists on the ***bacterial*** surface
 and free as a macromol. aggregate in the supernatant of spent culture
 medium. It is composed of a single (repeating) 16,000-dalton (D)
 polypeptide subunit. The AFA-I protein amino acid compn. is remarkable
 for the presence of 2.5-3.0 cysteines/subunit and for a marked decrease in
 hydrophobic amino acids as compared to subunits of E. coli pili. Since
 AFA-I travels as a monomer in SDS-PAGE under nonreducing conditions, no
 disulfide bonds exist between subunits and .gtoreq.1 free SH/subunit is
 available. The AFA-I N-terminal amino acid sequence through residue 24
 was unrelated to any known E. coli fimbrial sequence. Immuno-gold
 labeling demonstrated the afimbrial nature of the AFA-I protein on the
 bacterial cell surface. Anti-AFA-I sera bound AFA-I in Western
 blots of 4 of 16 X-binding E. coli urine isolates. They did not bind MS
 or P pili. HB 101 (pIL 14), the AFA-I recombinant strain, agglutinated
 only human or gorilla erythrocytes, indicating a preference for
 receptor mols. on the red cells of man and the anthropoid apes.
 AFA-I did not bind glycophorin A or sialyl glycosides and is therefore
 distinct from the E. coli X-binding adhesins with M and S specificity.
 The AFA-I ***receptor*** was found to be abundant and diffusely
 distributed on HeLa tissue culture monolayer cells surface by indirect
 fluorescent microscopy. Total lipid exts. of human erythrocytes and
 voided uroepithelial cells proved neg. or specific binding of AFA-I. The
 AFA-I protein was shown to bind to a doublet of probably peripheral
 (glyco)proteins from human erythrocyte ***ghosts*** of
 .apprx.96,000-98,000 mol. wt.
 TI Characterization and ***receptor*** binding specificities of the
 X-binding UTI Escherichia coli adhesin AFA-I
 AB . . . afimbrial E. coli adhesion was purified from a recombinant strain

and chem., functionally, and serol. characterized. AFA-I exists on the
 bacterial surface and free as a macromol. aggregate in the
 supernatant of spent culture medium. It is composed of a single. . .
 unrelated to any known E. coli fimbrial sequence. Immuno-gold labeling
 demonstrated the afimbrial nature of the AFA-I protein on the
 bacterial cell surface. Anti-AFA-I sera bound AFA-I in Western
 blots of 4 of 16 X-binding E. coli urine isolates. They did. . . P
 pili. HB 101 (pIL 14), the AFA-I recombinant strain, agglutinated only
 human or gorilla erythrocytes, indicating a preference for
 receptor mols. on the red cells of man and the anthropoid apes.
 AFA-I did not bind glycophorin A or sialyl glycosides and is therefore
 distinct from the E. coli X-binding adhesins with M and S specificity.
 The AFA-I ***receptor*** was found to be abundant and diffusely
 distributed on HeLa tissue culture monolayer cells surface by indirect
 fluorescent microscopy. Total. . . binding of AFA-I. The AFA-I
 protein was shown to bind to a doublet of probably peripheral
 (glyco)proteins from human erythrocyte ***ghosts*** of
 .apprx.96,000-98,000 mol. wt.

ST adhesion AFAI Escherichia ***receptor*** binding
 IT Escherichia coli
 (adhesin AFA-I of, characterization and ***receptor*** binding of)

IT Agglutinins and Lectins
 RL: BIOL (Biological study)
 (adhesive factors, AFA-I, of Escherichia coli, characterization and
 receptor binding of)

L7 ANSWER 46 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
 STN DUPLICATE 12

AN 1986:280942 BIOSIS <<LOGINID::20091202>>
 DN PREV198682024805; BA82:24805

TI THE BINDING OF PSEUDOMONAS-AERUGINOSA OUTER MEMBRANE ***GHOSTS*** TO
 HUMAN BUCCAL EPITHELIAL CELLS.

AU DOIG P [Reprint author]; FRANKLIN A L; IRVIN R T
 CS DEP BOTANY MICROBIOL, ERINDALE COLL, UNIV TORONTO, MISSISSAUGA, ONT, CAN
 L5L 1C6

SO Canadian Journal of Microbiology, (1986) Vol. 32, No. 2, pp. 160-166.
 CODEN: CJMIAZ. ISSN: 0008-4166.

DT Article
 FS BA
 LA ENGLISH

ED Entered STN: 4 Jul 1986
 Last Updated on STN: 4 Jul 1986

AB The binding of outer membrane (OM) ***ghosts*** derived from
 Pseudomonas aeruginosa strain 492c to human buccal epithelial cells (BECs)
 was examined. Electron microscopic examination of the binding of OM
 ghosts to BECs revealed direct OM ***ghost*** -BEC
 interaction.

Equilibrium analysis of the binding of OM ***ghosts*** to trypsinized
 BECs employing the Langmuir adsorption isotherm indicated the number of
 binding sites (N) to be 1.3 .times. 10⁻⁴ .mu.g protein per BEC with an
 apparent association constant (Ka) of 3.4 .times. 10⁻² mL/.mu.g protein.
 The Langmuir analysis of binding of OM ***ghosts*** to untrypsinized
 BECs was complex, suggesting two possible classes of receptors, a high
 affinity-low copy number class (Ka, 7.8 .times. 10⁻² mL/.mu.g protein; N,
 8.6 .times. 10⁻⁵ .mu.g protein per BEC) and a low affinity-high copy
 number class (Ka, 3.7 .times. 10⁻³ mL/.mu.g protein; N, 9.2 .times. 10⁻⁴
 .mu.g protein per BEC). Sugar inhibition studies incorporating

D-galactose enhanced binding to each BEC type. N-Acetylneuraminic acid and N-acetylglucosamine both enhanced binding of OM ***ghosts*** to untrypsinized BECs, while inhibiting binding to trypsinized BECs. D-Arabinose inhibited binding to both BEC types. Binding of OM ***ghosts*** to both BEC types was greatly inhibited by D-fucose, while L-fucose only greatly inhibited binding to untrypsinized BECs. These sugar inhibition data demonstrated a difference in the binding of OM ***ghosts*** to trypsinized and untrypsinized BECs and possibly reveal the nature of ***receptor*** (s), free of possible ***bacterial*** metabolic effects. These data indicated that OM ***ghosts*** from 492c appear to bind to BECs in a similar manner to the intact ***bacteria*** and represent a simple model system to study the adhesion of *P. aeruginosa* to BECs.

TI THE BINDING OF PSEUDOMONAS-AERUGINOSA OUTER MEMBRANE ***GHOSTS*** TO HUMAN BUCCAL EPITHELIAL CELLS.

AB The binding of outer membrane (OM) ***ghosts*** derived from *Pseudomonas aeruginosa* strain 492c to human buccal epithelial cells (BECs) was examined. Electron microscopic examination of the binding of OM ***ghosts*** to BECs revealed direct OM ***ghost***-BEC interaction.

Equilibrium analysis of the binding of OM ***ghosts*** to trypsinized BECs employing the Langmuir adsorption isotherm indicated the number of binding sites (N) to be 1.3×10^{-4} . . . BEC with an apparent association constant (K_a) of 3.4×10^{-2} mL/.μg protein. The Langmuir analysis of binding of OM ***ghosts*** to untrypsinized BECs was complex, suggesting two possible classes of receptors, a high affinity-low copy number class (K_a, 7.8×10^{-2} mL/.μg protein). Sugar inhibition studies incorporating D-galactose enhanced binding to each BEC type. N-Acetylneuraminic acid and N-acetylglucosamine both enhanced binding of OM ***ghosts*** to untrypsinized BECs, while inhibiting binding to trypsinized BECs. D-Arabinose inhibited binding to both BEC types. Binding of OM ***ghosts*** to both BEC types was greatly inhibited by D-fucose, while L-fucose only greatly inhibited binding to untrypsinized BECs. These sugar inhibition data demonstrated a difference in the binding of OM ***ghosts*** to trypsinized and untrypsinized BECs and possibly reveal the nature of ***receptor*** (s), free of possible ***bacterial*** metabolic effects. These data indicated that OM ***ghosts*** from 492c appear to bind to BECs in a similar manner to the intact ***bacteria*** and represent a simple model system to study the adhesion of *P. aeruginosa* to BECs.

ORGN Classifier

Pseudomonadaceae 06508

Super Taxa

Gram-Negative Aerobic Rods and Cocci; Eubacteria; ***Bacteria*** ; Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

L7 ANSWER 47 OF 68 LIFESCI COPYRIGHT 2009 CSA on STN

AN 86:72100 LIFESCI <<LOGINID::20091202>>

TI Studies on the staphylococcal alpha-toxin ***receptor*** on myelin and RB-RBC.

BACTERIAL PROTEIN TOXINS.

AU Harshman, S.; Sugg, N.; Falmagne, P. [editor]; Alouf, J.E. [editor]; Fehrenbach, F.J. [editor]; Jeljaszewicz, J. [editor]; Thelestam, M.

[editor]
 CS Dep. Microbiol., Vanderbilt Univ. Sch. Med., Nashville, TN 37232, USA
 SO ZENTRALBL. BAKTERIOL. MIKROBIOL. HYG., (1986) pp. 213-220.
 Meeting Info.: 2. European Workshop on Bacterial Protein Toxins. Wepion
 (Belgium). 30 Jun-4 Jul 1985.
 ISBN: 3-437-11083-7.
 DT Book
 TC Conference
 FS J; M
 LA English
 AB Staphylococcal alpha-toxin is an extracellular protein that is produced by
 most pathogenic strains of Staphylococcus aureus . It is selectively
 hemolytic, induces spastic paralysis in smooth muscle, provokes dermal
 necrosis, and is lethal for most laboratory animals. Although the detailed
 molecular mechanism of its lethal activity is not known, several lines of
 evidence led to the conclusion that the central or peripheral nervous
 tissue is the critical target organ. Both Schwann cell generated myelin of
 peripheral tissue and oligodendroglial cell generated myelin of central
 nervous tissue are susceptible to selective disruption by alpha-toxin. The
 authors report here recent data that suggests that a common lipoprotein
 exists in the membranes of myelin and Rb-rbc ***ghosts*** that may
 function as the specific alpha-toxin ***receptor*** .
 TI Studies on the staphylococcal alpha-toxin ***receptor*** on myelin and
 RB-RBC.
 BACTERIAL PROTEIN TOXINS.
 AB . . . The authors report here recent data that suggests that a common
 lipoprotein exists in the membranes of myelin and Rb-rbc ***ghosts***
 that may function as the specific alpha-toxin ***receptor*** .

L7 ANSWER 48 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 1984:486921 CAPLUS <<LOGINID::20091202>>
 DN 101:86921
 OREF 101:13301a,13304a
 TI Specific binding assays utilizing analyte-cytolysin conjugates
 IN Freytag, William J.; Litchfield, William John
 PA du Pont de Nemours, E. I., and Co. , USA
 SO Eur. Pat. Appl., 48 pp.
 CODEN: EPXXDW
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	EP 106370	A2	19840425	EP 1983-110469	19831020
	EP 106370	A3	19860226		
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	US 4517303	A	19850514	US 1982-435455	19821020
	CA 1206899	A1	19860701	CA 1983-439211	19831018
	DK 8304811	A	19840421	DK 1983-4811	19831019
	JP 59094069	A	19840530	JP 1983-194473	19831019
PRAI	US 1982-435455	A	19821020		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB A rapid and sensitive membrane lytic assay is described for the detn. of
 low concns. (micromolar to picomolar range) of analytes (e.g., drugs,
 metabolites, hormones, pesticides, food toxins, viruses, cancer cell
 markers) in a liq. medium by using a new analyte deriv.-cytolysin
 conjugate, an analyte-specific binding agent (e.g., antibody, hormone

receptor , ***lectin*** , specific binding protein), and vesicles (e.g., lipid vesicles, erythrocytes, or their ***ghosts***) contg. detectable markers (enzymes, cofactors, chromophores, fluorophores, ions, spin labels). Uncombined conjugate alters the permeability of the vesicles, resulting in release and quantitation of the marker which is correlated to the concn. of analyte initially present. Thus, digoxin was detd. by a homogeneous immunoassay by using antidigoxin antibodies purified by affinity chromatog., ouabain-melittin conjugate, and lipid vesicles with sequestered alk. phosphatase. The absorbance of the soln. was monitored continuously at 410 nm.

OSC.G 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (22 CITINGS)

AB . . . cell markers) in a liq. medium by using a new analyte deriv.-cytolysin conjugate, an analyte-specific binding agent (e.g., antibody, hormone ***receptor*** , ***lectin*** , specific binding protein), and vesicles (e.g., lipid vesicles, erythrocytes, or their ***ghosts***) contg. detectable markers (enzymes, cofactors, chromophores, fluorophores, ions, spin labels). Uncombined conjugate alters the permeability of the vesicles, resulting in. . .

IT ***Bacteria***

(surface markers of, detn. of, by membrane lysis assay)

L7 ANSWER 49 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN DUPLICATE 13

AN 1984:317890 BIOSIS <<LOGINID::20091202>>

DN PREV198478054370; BA78:54370

TI PHYSIOLOGICAL RESPONSES OF BACILLUS SPECIES TO CONCANAVALIN A 1. BINDING OF CONCANAVALIN A TO BACILLUS-CEREUS ATCC-14579 AND BACILLUS-LICHENIFORMIS IFO-12107.

AU CHAN K-Y [Reprint author]; LAU T-M

CS DEP BIOL, CHIN UNIV HONG KONG, SHATIN, NT, HONG KONG

SO Microbios, (1984) Vol. 39, No. 156, pp. 121-128.

CODEN: MCBIA7. ISSN: 0026-2633.

DT Article

FS BA

LA ENGLISH

AB The binding of labeled concanavalin A (Con A), a mitogenic protein, to cells of B. cereus ATCC 14579 and B. licheniformis IFO 12107 indicated that almost identical levels of 3H-Con A were located in the whole cell, and the protoplast and the membrane ***ghost*** fractions, evidence that Con A did not bind to the teichoic acids and peptidoglycans of the cell walls, but only to the membrane teichoic acids. The binding of 3H-Con A to the cells was temperature-dependent. Greater amounts of H-Con A bound with various cell fractions at 35.degree. C than at 0.degree. C. Evidently, at 35.degree. C Con A not only bound to the specific ***receptor*** sites of the cells but also bound to the cell envelope

by

non-specific binding which occurred mainly on the surface of the ***bacterial*** cells. That only negligible levels of 3H-Con A bound with the cytoplasm fractions suggested that the cells were unable to transport Con A molecules into the cytoplasm.

AB. . . indicated that almost identical levels of 3H-Con A were located in the whole cell, and the protoplast and the membrane ***ghost*** fractions, evidence that Con A did not bind to the teichoic acids and peptidoglycans of the cell walls, but only. . . fractions at 35.degree. C than at 0.degree. C. Evidently, at 35.degree. C Con A not only bound to the specific ***receptor*** sites of the cells but also bound to the cell envelope by non-specific binding which occurred mainly on the

surface of the ***bacterial*** cells. That only negligible levels of 3H-Con A bound with the cytoplasm fractions suggested that the cells were unable to. . .

ORGN Classifier

Endospore-forming Gram-Positives 07810

Super Taxa

Eubacteria; ***Bacteria*** ; Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

L7 ANSWER 50 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1983:277110 BIOSIS <<LOGINID::20091202>>

DN PREV198376034602; BA76:34602

TI AGGREGATION OF HUMAN PLATELETS AND ADHESION OF STREPTOCOCCUS-SANGUIS.

AU HERZBERG M C [Reprint author]; BRINTZENHOFE K L; CLAWSON C C

CS SCH DENT, UNIV MINN, MINNEAPOLIS, MINN 55455, USA

SO Infection and Immunity, (1983) Vol. 39, No. 3, pp. 1457-1469.

CODEN: INFIBR. ISSN: 0019-9567.

DT Article

FS BA

LA ENGLISH

AB The hypothesis that human platelets selectively bind or adhere to strains of *S. Sanguis* and *S. mutans* and aggregate, as a result, into an in vitro thrombus was investigated. Adhesion was uncoupled from activation and aggregation by incubating streptococci with platelet ***ghosts*** in a simple, quantitative assay. Adhesion was mediated by protease-sensitive components on the streptococci and platelet ***ghosts*** rather than cell surface carbohydrates or dextrans, plasma components or divalent cations. The same streptococci were also studied by standard aggregometry techniques. Platelet-rich plasma was activated and aggregated by certain isolates of *S. sanguis*. Platelet ***ghosts*** bound the same strains selectively under Ca^{2+} and plasma-depleted conditions. Fresh platelets activated after washing; Ca^{2+} had to be restored. Aggregation required fresh platelets in Ca^{2+} -restored plasma and was inducible by washed streptococcal cell walls. These reactions in the binding and aggregometry assays were confirmed by transmission EM. Surface microfibrils on intact *S. sanguis* were identified. These appendages bound *S. sanguis* to platelets. The selectivity of adhesion of the various *S. sanguis* strains to platelet ***ghosts*** or Ca^{2+} - and plasma-depleted fresh washed platelets was similar for all donors. Thus, the platelet binding site was expressed widely in the population and was unlikely to be an artifact of membrane aging or preparation. Since selective adhesion of *S. sanguis* to platelets was apparently required for aggregation, functionally defined receptors for ligands on certain strains of *S. sanguis* may be present on human platelets. Some differences in the selectivity and rate of the aggregation response were noted among platelet donors; the meaning of the variability requires further study. These interactions may contribute to platelet accretion in the initiation and development of vegetative lesions in subacute ***bacterial*** endocarditis.

AB. . . result, into an in vitro thrombus was investigated. Adhesion was uncoupled from activation and aggregation by incubating streptococci with platelet ***ghosts*** in a simple, quantitative assay. Adhesion was mediated by protease-sensitive components on the streptococci and platelet ***ghosts*** rather than cell surface carbohydrates or dextrans, plasma components or divalent cations. The same streptococci were also studied by standard aggregometry techniques. Platelet-rich plasma was activated

and aggregated by certain isolates of *S. sanguis*. Platelet ***ghosts*** bound the same strains selectively under Ca²⁺ and plasma-depleted conditions. Fresh platelets activated after washing; Ca²⁺ had to be restored.. . . identified. These appendages bound *S. sanguis* to platelets. The selectivity of adhesion of the various *S. sanguis* strains to platelet ***ghosts*** or Ca²⁺- and plasma-depleted fresh washed platelets was similar for all donors. Thus, the platelet binding site was expressed widely. . . requires further study. These interactions may contribute to platelet accretion in the initiation and development of vegetative lesions in subacute ***bacterial*** endocarditis.

IT Miscellaneous Descriptors

STREPTOCOCCUS-MUTANS HUMAN PLATELET ***GHOSTS*** PROTEASE SENSITIVE
COMPONENTS CALCIUM ION DEPLETION PLASMA DEPLETION SURFACE MICRO FIBRILS
LIGAND RECEPTORS ACCRETION VEGETATIVE LESIONS SUBACUTE
BACTERIAL ENDO CARDITIS

ORGN Classifier

Gram-Positive Cocci 07700
Super Taxa
Eubacteria; ***Bacteria*** ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates,. . .

L7 ANSWER 51 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN

AN 1983:245983 BIOSIS <<LOGINID::20091202>>

DN PREV198376003475; BA76:3475

TI MICRO INJECTION OF MACRO MOLECULES INTO NORMAL MURINE LYMPHOCYTES BY MEANS
OF CELL FUSION 2. ENHANCEMENT AND SUPPRESSION OF MITOGENIC RESPONSES BY
MICRO INJECTION OF MONO CLONAL ANTI CYCLIC AMP INTO B LYMPHOCYTES.

AU OHARA J [Reprint author]; SUGI M; FUJIMOTO M; WATANABE T

CS DEP OF IMMUNOL, SAGA MED SCH, NABESHIMA, SAGA 840-01, JPN

SO Journal of Immunology, (1982) Vol. 129, No. 3, pp. 1227-1232.
CODEN: JOIMA3. ISSN: 0022-1767.

DT Article

FS BA

LA ENGLISH

AB Reproducible methods are now available for introducing protein molecules such as antibodies into normal murine lymphocytes by fusion with protein molecule-containing erythrocyte ***ghosts***. Monoclonal antibodies against cAMP were raised by hybridoma technique and packed into erythrocyte ***ghosts***. Then, monoclonal anti-cAMP containing ***ghosts*** were fused with splenic B lymphocytes by polyethylene glycol-mediated fusion at various intervals after LPS stimulation. This method made it possible to quantitatively microinject antibodies into B lymphocytes. Microinjection of anti-cAMP antibody molecules into lymphocytes at a very early stage of LPS [lipopolysaccharide] stimulation resulted in a marked enhancement of DNA synthetic responses and increased numbers of plaque-forming cells. Intracellular cAMP levels were markedly decreased after microinjection of monoclonal anti-cAMP, suggesting that lowering the intracellular cAMP level in the B lymphocytes at an early stage of stimulation might have induced the enhanced proliferative and

differentiative responses to LPS. Similar enhancing effects on cell proliferation were obtained when antibodies were injected 18 h after stimulation. Microinjection of anti-cAMP at 12 h after culture inhibited the DNA synthetic responses, and induction of plaque-forming cells was suppressed when anti-cAMP was injected 6 h after LPS stimulation. The data suggest the biphasic regulatory roles of cAMP at the early stage of B lymphocyte activation. This approach may be useful in identifying regulatory molecules in B lymphocytes induced by mitogenic or antigenic stimulation.

AB. . . are now available for introducing protein molecules such as antibodies into normal murine lymphocytes by fusion with protein molecule-containing erythrocyte ***ghosts***. Monoclonal antibodies against cAMP were raised by hybridoma technique and packed into erythrocyte ***ghosts***. Then, monoclonal anti-cAMP containing ***ghosts*** were fused with splenic B lymphocytes by polyethylene glycol-mediated fusion at various intervals after LPS stimulation. This method made it. . .

IT Miscellaneous Descriptors

PLAQUE FORMING CELL LIPO POLY ***SACCHARIDE*** DNA SYNTHESIS

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .

L7 ANSWER 52 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1982:237843 BIOSIS <<LOGINID::20091202>>

DN PREV198274010323; BA74:10323

TI MICRO INJECTION OF MACRO MOLECULES INTO NORMAL MURINE LYMPHOCYTES BY CELL FUSION TECHNIQUE 1. QUANTITATIVE MICRO INJECTION OF ANTIBODIES INTO NORMAL SPLENIC LYMPHOCYTES.

AU OHARA J [Reprint author]; WATANABE T

CS DEP IMMUNOL, SAGA MED SCH, NABESHIMA, SAGA 840-01, JAPAN

SO Journal of Immunology, (1982) Vol. 128, No. 3, pp. 1090-1096.

CODEN: JOIMA3. ISSN: 0022-1767.

DT Article

FS BA

LA ENGLISH

AB Human erythrocyte ***ghosts*** loaded with various kinds of protein molecules were fused with mouse splenic lymphocytes by means of polyethylene glycol supplemented with poly-L-arginine and dimethylsulfoxide. This fusion method made quantitative microinjection of IgG and other proteins into intact lymphocytes possible. The injection itself did not alter cell viability and lymphocytes given protein molecules retained intact response activity when they were stimulated with mitogens. Rabbit anti-cAMP was purified by affinity chromatography and injected into lymphocytes. Antibody activity in the cell lysates was measured by using 125I-labeled cAMP as an antigen and it was shown that antibody molecules were quantitatively injected and immunologically active

in the cells. Antigen binding activity of anti-cAMP antibodies in the nonstimulated lymphocytes was stable and intact even 24 h after microinjection, whereas the activity rapidly decreased in mitogen-stimulated lymphocytes, indicating that some immunologic or enzymatic mechanisms for inactivating antibodies were induced in mitogen-stimulated cells. Microinjection of anti-cAMP markedly enhanced the proliferative responses of lymphocytes to mitogens such as concanavalin A or lipopolysaccharide and reversed the effect of a known elevator of intracellular cAMP. These observations have implications for the role of cAMP in early lymphocyte activation events.

AB Human erythrocyte ***ghosts*** loaded with various kinds of protein molecules were fused with mouse splenic lymphocytes by means of polyethylene glycol supplemented with. . .

IT Miscellaneous Descriptors
HUMAN ERYTHROCYTE ***GHOST*** RABBIT ANTIGEN BINDING ACTIVITY
LYMPHOCYTE ACTIVATION CYCLIC AMP CONCAVALIN A LIPO POLY
SACCHARIDE

ORGN Classifier
Bacteria 05000
Super Taxa
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

ORGN Classifier
Leguminosae 26260
Super Taxa
Dicotyledones; Angiospermae; Spermatophyta; Plantae
Taxa Notes
Angiosperms, Dicots, Plants, Spermatophytes, Vascular Plants

L7 ANSWER 53 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 14

AN 1982:198350 BIOSIS <<LOGINID::20091202>>
DN PREV198273058334; BA73:58334
TI FAST RESPONSES OF ***BACTERIAL*** MEMBRANES TO VIRUS ADSORPTION A
FLUORESCENCE STUDY.

AU BAYER M E [Reprint author]; BAYER M H
CS INST FOR CANCER RES, FOX CHASE CANCER CENT, PHILADELPHIA, PA 19111, USA
SO Proceedings of the National Academy of Sciences of the United States of
America, (1981) Vol. 78, No. 9, pp. 5618-5622.
CODEN: PNASA6. ISSN: 0027-8424.

DT Article
FS BA
LA ENGLISH

AB After collision with their host cells, virus particles may remain mobile on cell surfaces until they become attached at firm binding sites. It is proposed that a virion will arrive within a typical median time at such a site, generating a membrane signal such as an increased membrane fluorescence in cells labeled with the voltage-sensitive dyes 8-anilino-1-naphthalene-sulfonate (Mg-salt) (ANS), N-phenylnaphthylamine (NPA) or 3,3'-dipentyl-2,2'-oxacarbocyanine (di-O-C5[3]). The time span between virus adsorption and fluorescence response varies widely among phages and also depends on ***bacterial*** [Escherichia coli, Salmonella ado] strain, metabolic state and type of dye. di-O-C5[3]-labeled cells react within 1 s to uncouplers such as carbonyl cyanide m-chlorophenylhydrazone (CCCP). Cells labeled with ANS and NPA react to CCCP in 4-6 s. ***Bacteriophages*** (T4, T5, .chi., and

BF23), added to ANS-labeled cells, change the fluorescence in 9-15 s. T-even ***ghosts*** cause a response at identical times. Baseplate-defective phage mutant T412- and isolated adsorption organelles of smaller viruses fail to cause an effect. di-O-C5[3]-labeled cells respond to T4 at a multiplicity of infection .gtoreq. 40 within 1 s. A longer time (8 s) is required at lower multiplicities. The ***receptor*** -degrading phages .epsilon.15, .epsilon.34, c341 and K29 need the longest time (1 min for ANS) to cause a fluorescence increase. The delayed fluorescence response may be concomitant with the surface walk of the virion which is terminated at an injection site. T4 tail sheath contraction coincided with the onset of the membrane fluorescence response.

TI FAST RESPONSES OF ***BACTERIAL*** MEMBRANES TO VIRUS ADSORPTION A FLUORESCENCE STUDY.

AB. . . or 3,3'-dipentyl-2,2'-oxacarbocyanine (di-O-C5[3]). The time span between virus adsorption and fluorescence response varies widely among phages and also depends on ***bacterial*** [Escherichia coli, Salmonella ado] strain, metabolic state and type of dye. di-O-C5[3]-labeled cells react within 1 s to uncouplers such as carbonyl cyanide m-chlorophenylhydrazine (CCCP). Cells labeled with ANS and NPA react to CCCP in 4-6 s. ***Bacteriophages*** (T4, T5, .chi., and BF23), added to ANS-labeled cells, change the fluorescence in 9-15 s. T-even ***ghosts*** cause a response at identical times. Baseplate-defective phage mutant T412- and isolated adsorption organelles of smaller viruses fail to cause. . . a multiplicity of infection .gtoreq. 40 within 1 s. A longer time (8 s) is required at lower multiplicities. The ***receptor*** -degrading phages .epsilon.15, .epsilon.34, c341 and K29 need the longest time (1 min for ANS) to cause a fluorescence increase. The. . .

IT Miscellaneous Descriptors
 ESCHERICHIA-COLI SALMONELLA-ADO PHAGE T-4 PHAGE T-5 T EVEN PHAGE
 GHOSTS PHAGE CHI PHAGE BF-23 ***RECEPTOR*** DEGRADING
 PHAGE
 EPSILON 15 PHAGE EPSILON 34 PHAGE C-341 PHAGE K-29 8 ANILINO-1
 NAPHTHALENESULFONATE N PHENYL NAPHTHYLAMINE 3 3' DI. . .

ORGN . . .
 Viruses; Microorganisms
 Taxa Notes
 Double-Stranded DNA Viruses, Microorganisms, Viruses

ORGN Classifier
 Enterobacteriaceae 06702
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Hepaticae 21400
 Super Taxa
 Bryophyta; Plantae
 Taxa Notes
 Bryophytes, Nonvascular Plants, Plants

L7 ANSWER 54 OF 68 MEDLINE on STN
 AN 1981266179 MEDLINE <<LOGINID::20091202>>
 DN PubMed ID: 6790668
 TI Adsorption of the defective phage PBS Z1 to Bacillus subtilis 168 Wt.

AU Steensma H Y
SO The Journal of general virology, (1981 Jan) Vol. 52, No. Pt 1, pp. 93-101.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198110
ED Entered STN: 16 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 25 Oct 1981

AB Three aspects of the adsorption of the defective phage PBS Z1 to *Bacillus subtilis* 168 Wt have been investigated. These are the kinetics, the number of receptors on the cell wall and the character of these receptors. The reaction constants for the binding of phages onto receptors, for the dissociation of the phage- ***receptor*** complex and for the transition from reversible to irreversible binding of the phages were calculated from adsorption curves obtained by an enzyme-linked immunosorbent assay (ELISA). They were 1.8×10^{-13} , 6.7×10^{-2} and 9.0×10^{-3} respectively. The maximum number of phages adsorbed per cell was 2700, a number limited by the surface area of the cells. Apart from the receptors on the cell wall, receptors on the cell membrane were found. This was concluded from additional adsorption experiments with stable L-forms and contracted phages. Based on these results, together with data from the literature on ***bacteriocins***, phage ***ghosts*** and yeast killer factors, a hypothesis concerning the first stage of killing by defective phages has been formulated.

AB . . . the character of these receptors. The reaction constants for the binding of phages onto receptors, for the dissociation of the phage- ***receptor*** complex and for the transition from reversible to irreversible binding of the phages were calculated from adsorption curves obtained by. . . additional adsorption experiments with stable L-forms and contracted phages. Based on these results, together with data from the literature on ***bacteriocins***, phage ***ghosts*** and yeast killer factors, a hypothesis concerning the first stage of killing by defective phages has been formulated.

CT Adsorption
*Bacillus subtilis: ME, metabolism
****Bacteriophages: ME, metabolism****
Cell Membrane: AN, analysis
Cell Wall: AN, analysis
Kinetics
Receptors, Virus: AN, analysis
*Receptors, Virus: ME, metabolism

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AN 1979:247556 BIOSIS <<LOGINID::20091202>>
DN PREV197968050060; BA68:50060

TI STRUCTURAL AND BIOCHEMICAL EXAMINATION OF ***GHOSTS*** DERIVED FROM A DEEP ROUGH HEPTOSE DEFICIENT LIPO POLY ***SACCHARIDE*** STRAIN AND A SMOOTH STRAIN OF *ESCHERICHIA-COLI*.

AU IRVIN R T [Reprint author]; LAM J; COSTERTON J W
CS DEP BIOL, UNIV CALGARY, CALGARY, ALBERTA T2N 1N4, CAN
SO Canadian Journal of Microbiology, (1979) Vol. 25, No. 4, pp. 436-446.
CODEN: CJMIAZ. ISSN: 0008-4166.
DT Article

FS BA
 LA ENGLISH
 AB Outer membrane derived ***ghosts*** can be readily generated from smooth and deep rough (heptose-deficient LPS [lipopolysaccharide]) strains of E. coli 08. Morphological and biochemical studies confirmed that ***ghosts*** of both strains are composed of protein (4 major proteins), LPS and phospholipid (cardiolipin and phosphatidylethanolamine) in the form of a single membrane of roughly the same shape as intact normal cells. The ***ghost*** membrane cleaves only slightly in freeze-etch preparations of ***ghosts*** derived from the smooth strain as compared to the extensive cleavage plane of ***ghosts*** derived from the rough strain. The asymmetrical distribution of ***ghost*** proteins was visualized, by critical point drying and shadowing with Pt, as a relatively smooth outer surface with some discernible particles (10-15 nm) and an extremely particulate inner surface (10-15 nm particles). ***Ghosts*** derived from the smooth strain retained their structure following chloroform-methanol extraction, while ***ghosts*** derived from the rough strain fragmented with chloroform-methanol extraction. LPS-protein interactions and protein-protein interactions are apparently significant in maintaining the ***ghost*** structure.

TI STRUCTURAL AND BIOCHEMICAL EXAMINATION OF ***GHOSTS*** DERIVED FROM A DEEP ROUGH HEPTOSE DEFICIENT LIPO POLY ***SACCHARIDE*** STRAIN AND A SMOOTH STRAIN OF ESCHERICHIA-COLI.

AB Outer membrane derived ***ghosts*** can be readily generated from smooth and deep rough (heptose-deficient LPS [lipopolysaccharide]) strains of E. coli 08. Morphological and biochemical studies confirmed that ***ghosts*** of both strains are composed of protein (4 major proteins), LPS and phospholipid (cardiolipin and phosphatidylethanolamine) in the form of a single membrane of roughly the same shape as intact normal cells. The ***ghost*** membrane cleaves only slightly in freeze-etch preparations of ***ghosts*** derived from the smooth strain as compared to the extensive cleavage plane of ***ghosts*** derived from the rough strain. The asymmetrical distribution of ***ghost*** proteins was visualized, by critical point drying and shadowing with Pt, as a relatively smooth outer surface with some discernible particles (10-15 nm) and an extremely particulate inner surface (10-15 nm particles). ***Ghosts*** derived from the smooth strain retained their structure following chloroform-methanol extraction, while ***ghosts*** derived from the rough strain fragmented with chloroform-methanol extraction. LPS-protein interactions and protein-protein interactions are apparently significant in maintaining the ***ghost*** structure.

ORGN Classifier
 Enterobacteriaceae 06702
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 56 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
 AN 1979:215069 BIOSIS <<LOGINID::20091202>>
 DN PREV197968017573; BA68:17573

TI BETA ADRENERGIC ***RECEPTOR*** AGONISTS INCREASE PHOSPHO LIPID
 METHYLATION MEMBRANE FLUIDITY AND BETA ADRENERGIC ***RECEPTOR***
 ADENYLATE CYCLASE EC-4.6.1.1 COUPLING.
 AU HIRATA F [Reprint author]; STRITTMATTER W J; AXELROD J
 CS LAB CLIN SCI, NATL INST MENT HEALTH, BETHESDA, MD 20014, USA
 SO Proceedings of the National Academy of Sciences of the United States of
 America, (1979) Vol. 76, No. 1, pp. 368-372.
 CODEN: PNASA6. ISSN: 0027-8424.
 DT Article
 FS BA
 LA ENGLISH
 AB The .beta.-adrenergic agonist L-isoproterenol stimulated the enzymic
 synthesis of phosphatidyl-N-monomethylethanolamine and phosphatidylcholine
 in rat reticulocyte ***ghosts*** containing the methyl donor
 S-adenosyl-L-methionine. The stimulation was stereospecific,
 dose-dependent, and inhibited by .beta.-adrenergic agonist propranolol.
 The addition of GTP inside the resealed ***ghosts*** shifted the
 dose-response of phospholipid methylation by L-isoproterenol to the left
 by 2 orders of magnitude. Direct stimulation of adenylate cyclase [ATP
 pyrophosphate-lyase (cyclizing), EC 4.6.1.1] with NaF or cholera toxin did
 not increase the methylation of phospholipids. At a concentration of
 S-adenosyl-L-methionine that stimulates synthesis of
 phosphatidyl-N-monomethylethanolamine, the activity of
 sioproterenol-sensitive adenylate cyclase was increased 2-fold without
 changes in the basal activity of adenylate cyclase and the number of
 .beta.-adrenergic receptors. The increase of phospholipid methylation by
 L-isoproterenol decreased membrane viscosity and increased translocation
 of methylated lipids. These findings indicate that enhancement of
 phospholipid methylation by L-isoproterenol decreases membrane
 microviscosity and thus increases lateral movement of the
 .beta.-adrenergic receptors and coupling with adenylate cyclase.
 TI BETA ADRENERGIC ***RECEPTOR*** AGONISTS INCREASE PHOSPHO LIPID
 METHYLATION MEMBRANE FLUIDITY AND BETA ADRENERGIC ***RECEPTOR***
 ADENYLATE CYCLASE EC-4.6.1.1 COUPLING.
 AB The .beta.-adrenergic agonist L-isoproterenol stimulated the enzymic
 synthesis of phosphatidyl-N-monomethylethanolamine and phosphatidylcholine
 in rat reticulocyte ***ghosts*** containing the methyl donor
 S-adenosyl-L-methionine. The stimulation was stereospecific,
 dose-dependent, and inhibited by .beta.-adrenergic agonist propranolol.
 The addition of GTP inside the resealed ***ghosts*** shifted the
 dose-response of phospholipid methylation by L-isoproterenol to the left
 by 2 orders of magnitude. Direct stimulation of adenylate. . .
 ORGN Classifier
 Vibrionaceae 06704
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms
 ORGN Classifier
 Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Taxa Notes
 Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .

STN
AN 1979:265819 BIOSIS <<LOGINID::20091202>>
DN PREV197968068323; BA68:68323
TI EFFECT OF FLUORIDE AND 5 GUANYLYL IMIDO DI PHOSPHATE ON CHOLERA TOXIN TREATED CELL.
AU GANGULY U [Reprint author]; GREENOUGH W B III
CS CHOLER RES CENT, CALCUTTA 700 016, W BENGAL, INDIA
SO Indian Journal of Experimental Biology, (1978) Vol. 16, No. 12, pp. 1271-1273.
CODEN: IJEBA6. ISSN: 0019-5189.
DT Article
FS BA
LA ENGLISH
AB Cholera toxin stimulates [rat] fat cell adenylate cyclase after binding to a specific ganglioside (GM1) ***receptor*** . Epinephrine stimulates fat cell adenylate cyclase and this response is enhanced by prior exposure of cells to cholera toxin. Adenylate cyclase is stimulated by F- in a different way from hormones or cholera toxin. F- decreased the response to toxin and epinephrine separately and together at concentrations of more than 1 mM. At 25.degree. C the direct stimulating effects of F- are minimal and the blocking action is manifest. 5'-Guanylyl-imidodiphosphate (Gpp(NH)p) caused no further stimulation of basal or epinephrine-responsive adenylate cyclase in toxin-pre-treated fat cell ***ghosts*** . Since F- blocks stimulation by epinephrine and cholera toxin, a common pathw4y is shared despite differing receptors. Cells treated with cholera toxin or Gpp(NH)p enhance response to epinephrine and cells pre-treated with toxin do not respond further to Gpp(NH)p, suggesting a shared regulator pathway between toxin and a guanylyl nucleotide.
AB Cholera toxin stimulates [rat] fat cell adenylate cyclase after binding to a specific ganglioside (GM1) ***receptor*** . Epinephrine stimulates fat cell adenylate cyclase and this response is enhanced by prior exposure of cells to cholera toxin. Adenylate. . . blocking action is manifest. 5'-Guanylyl-imidodiphosphate (Gpp(NH)p) caused no further stimulation of basal or epinephrine-responsive adenylate cyclase in toxin-pre-treated fat cell ***ghosts*** . Since F- blocks stimulation by epinephrine and cholera toxin, a common pathw4y is shared despite differing receptors. Cells treated with. . .
ORGN Classifier
Vibrionaceae 06704
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria ; Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .
L7 ANSWER 58 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
AN 1979:149053 BIOSIS <<LOGINID::20091202>>
DN PREV197967029053; BA67:29053
TI ROLE OF CHOLESTEROL IN THE ACTION OF CEREOLYSIN ON MEMBRANES.

AU COWELL J L [Reprint author]; BERNHEIMER A W
 CS DIV BACT PROD, BUR BIOL, FOOD DRUG ADM, BETHESDA, MD 20014, USA
 SO Archives of Biochemistry and Biophysics, (1978) Vol. 190, No. 2, pp. 603-610.
 CODEN: ABBIA4. ISSN: 0003-9861.

DT Article
 FS BA
 LA ENGLISH

AB The following evidence supports the concept that cholesterol in membranes is the ***receptor*** and target site for the cytolytic action of [Bacillus cereus] cereolysin. Of the various phospholipids, gangliosides and steroids tested, only cholesterol and closely related sterols (sitosterol and dihydrocholesterol) significantly inhibited the hemolytic activity of cereolysin. Acholeplasma laidlawii cells grown in the presence of cholesterol inhibited the hemolytic activity of cereolysin, but A. laidlawii grown in the absence of cholesterol did not. Incubation of A. laidlawii cells, grown in the absence of cholesterol, with a cholesterol-Tween 80 mixture reestablished the ability of the cells to bind cereolysin. Treatment of erythrocyte membranes and A. laidlawii cells containing cholesterol with cholesterol oxidase (EC 1.1.3.6, Brevibacterium sp.) abolished the ability of these membranes to bind cereolysin and inhibit the hemolytic activity of the toxin. Cereolysin could bind to and alter the permeability of both right-side-out ***ghosts*** and inside-out vesicles prepared from human erythrocytes, in agreement with other data that cholesterol is present on both sides of the erythrocyte membrane. Cereolysin caused the release of [14C]glucose from liposomes containing cholesterol, and this release was dependent on the amount of cholesterol in the liposomes.

AB The following evidence supports the concept that cholesterol in membranes is the ***receptor*** and target site for the cytolytic action of [Bacillus cereus] cereolysin. Of the various phospholipids, gangliosides and steroids tested, only. . . cereolysin and inhibit the hemolytic activity of the toxin. Cereolysin could bind to and alter the permeability of both right-side-out ***ghosts*** and inside-out vesicles prepared from human erythrocytes, in agreement with other data that cholesterol is present on both sides of. . .

ORGN Classifier
 Enterobacteriaceae 06702
 Super Taxa
 Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
 Bacteria ; Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Irregular Nonsporing Gram-Positive Rods 08890
 Super Taxa
 Actinomycetes and Related Organisms; Eubacteria; ***Bacteria*** ;
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Acholeplasmataceae 07511
 Super Taxa
 Mycoplasmatales; Mycoplasmas; Eubacteria; ***Bacteria*** ;
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 59 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
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AN 1978:140550 BIOSIS <<LOGINID::20091202>>

DN PREV197865027550; BA65:27550

TI EFFECTS OF CHOLERA ENTERO TOXIN ON CATECHOLAMINE STIMULATED CHANGES IN
CATION FLUXES CELL VOLUME AND CYCLIC AMP LEVELS IN THE TURKEY ERYTHROCYTE.

AU RUDOLPH S A [Reprint author]; SCHAFER D E; GREENGARD P

CS DEP PHARMACOL, SCH MED, CASE WEST RESERVE UNIV, CLEVELAND, OHIO 44106, USA

SO Journal of Biological Chemistry, (1977) Vol. 252, No. 20, pp. 7132-7139.
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

FS BA

LA ENGLISH

AB Treatment of turkey erythrocytes with cholera enterotoxin [from *Vibrio cholerae*] caused increases in basal cyclic[c]AMP levels, in the sensitivity of cAMP levels to .beta.-adrenergic stimulation and in the maximum level of cAMP attainable with catecholamines. These responses were first detected in the period from 30-60 min after addition of toxin. Na and K influxes in toxin-treated cells showed increases in basal levels and in sensitivity to catecholamines, but not in the maximal response attainable with catecholamines. Toxin-treated cells also exhibited a slow net uptake of water. In medium containing 15 mM K+, the enterotoxin caused a progressive decrease in the maximal catecholamine effect on cation fluxes; this decrease was not observed when the medium contained 2.5 mM K+. A net uptake of K+, Na+ and H2O, similar to that which occurs in response to catecholamines or cAMP at high extracellular K+ concentrations, also occurs with cholera enterotoxin; this accumulation of water and cations apparently has some feedback effect on cation fluxes, rendering them insensitive to cAMP. Cholera enterotoxin did not appear to affect the hormone- ***receptor*** interaction as judged by the binding of [3H]alprenolol, a .beta.-adrenergic antagonist. ***Ghosts*** prepared from control and toxin-treated erythrocytes had similar capacities and affinities for this ***ligand***. On the basis of the [3H]alprenolol binding data and the effect of various concentrations of isoproterenol on cation fluxes, normal cells appear to require occupancy of about 10 receptors to activate the cAMP-dependent flux mechanism, but in toxin-treated cells, occupancy of a single ***receptor*** appears to be sufficient. Cholera enterotoxin probably causes an alteration in the ***receptor*** -adenylate cyclase interaction and an increase in basal adenylylase activity. The effects of cholera enterotoxin on cation fluxes and volume changes in the turkey erythrocyte appear to be accounted for by these effects on cAMP accumulation.

AB. . . has some feedback effect on cation fluxes, rendering them insensitive to cAMP. Cholera enterotoxin did not appear to affect the hormone- ***receptor*** interaction as judged by the binding of [3H]alprenolol, a .beta.-adrenergic antagonist. ***Ghosts*** prepared from control and toxin-treated erythrocytes had similar capacities and affinities for this ***ligand***. On the basis of the [3H]alprenolol binding data and the effect of various concentrations of isoproterenol on cation fluxes, normal. . . require occupancy of about 10 receptors to activate the cAMP-dependent flux mechanism, but in toxin-treated cells, occupancy of a single ***receptor*** appears to be sufficient. Cholera enterotoxin probably causes an alteration in the ***receptor*** -adenylate cyclase interaction and an increase in basal adenylylase activity. The effects of cholera enterotoxin on cation fluxes and volume.

. .

ORGN Classifier
 Bacteria 05000
 Super Taxa
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

ORGN Classifier
 Galliformes 85536
 Super Taxa
 Aves; Vertebrata; Chordata; Animalia
 Taxa Notes
 Animals, Birds, Chordates, Nonhuman Vertebrates, Vertebrates

L7 ANSWER 60 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN
AN 1977:49238 BIOSIS <<LOGINID::20091202>>
DN PREV197713049238; BR13:49238
TI STRUCTURAL EXAMINATION OF CELL WALL ***GHOSTS*** OF A DEEP ROUGH AND A
SMOOTH STRAIN OF ESCHERICHIA-COLI 08.
AU IRVIN R T; LAM J; COSTERTON J W
SO Abstracts of the Annual Meeting of the American Society for Microbiology,
(1977) Vol. 77, pp. 182.
CODEN: ASMACK. ISSN: 0094-8519.
DT Article
FS BR
LA Unavailable
TI STRUCTURAL EXAMINATION OF CELL WALL ***GHOSTS*** OF A DEEP ROUGH AND A
SMOOTH STRAIN OF ESCHERICHIA-COLI 08.
IT Miscellaneous Descriptors
 ABSTRACT MEMBRANE PROTEINS LIPO POLY ***SACCHARIDE***

ORGN Classifier
 Bacteria 05000
 Super Taxa
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 61 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
STN DUPLICATE 15
AN 1977:144449 BIOSIS <<LOGINID::20091202>>
DN PREV197763039313; BA63:39313
TI LIPOLYTIC ACTION OF CHOLERA TOXIN ON FAT CELLS REEXAMINATION OF THE
CONCEPT IMPLICATING GM-1 GANGLIOSIDE AS THE NATIVE MEMBRANE
RECEPTOR .
AU KANFER J N; CARTER T P; KATZEN H M
SO Journal of Biological Chemistry, (1976) Vol. 251, No. 23, pp. 7610-7619.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
FS BA
LA Unavailable
AB The possible role of galactosyl-N-acetylgalactosaminyl-[N-
acetylneuraminy]-galactosylglucosyl-ceramide (GM1) ganglioside in the
lipolytic activity of cholera toxin [CT] on isolated [rat] fat cells was
examined. Analyses of the ganglioside content and composition of intact
fat cells, their membranous ***ghosts*** and the total particulate
fraction of these cells indicate that
N-acetylneuraminylgalactosylglucosylceramide (GM3) represents the major

ganglioside, with substantial amounts of N-acetylgalactosaminyl-[N-acetylneuraminyl]-galactosylglucosylceramide (GM2) and smaller amounts of other higher homologues also present. Native GM1 was not detected in any of these preparations. Examination of the relative capacities of various exogenously added radiolabeled sphingolipids to bind to the cells indicated that GM2 and glucosylsphingosine were accumulated by the cells to extents comparable to GM1. Galactosylsphingosine and sulfatide also exhibited significant, although lesser, binding affinities for the cells. The adipocytes appeared to nonspecifically bind exogenously added GM1; saturation of binding sites for GM1 could not be observed up to the highest concentration tested (2 .times. 10⁻⁴ M), wherein about 7 .times. 10⁹ molecules were associated with the cells. Essentially all exogenously added GM1 was found bound to the plasma membrane ***ghost*** fraction. Investigation of the biological responses of the cells confirmed their sensitivities to CT and epinephrine-stimulated lipolysis, as well as the lag period displayed during the toxin's action. While the toxin's lipolytic activity can be enhanced by prior treatment of the fat cells with GM1, added GM1 enhanced only the subsequent rate, but not the extent, of toxin stimulated glycerol release (lipolysis) from the cells. The ability of GM1 to enhance the toxin's activity at saturating or low toxin concentrations was unconfirmed. The limited ability of added GM1 to enhance the toxin's activity appeared in a unique bell-shaped dose-response manner. The inability of high levels of GM1 to stimulate a dose of toxin that was ineffective on native cells suggests that the earlier reported ability of crude brain gangliosides to accomplish this was due to some component other than GM1 in the crude extract. While several glycosphingolipids and some other carbohydrate-containing substances that were tested lacked the ability to mimic the enhancing effect of GM1, 4-methylumbelliferyl-.beta.-D-galactoside exhibited an effect similar to, although less pronounced than, that of GM1. These findings do not support the earlier hypotheses that GM1 is CT's naturally occurring membrane ***receptor*** on native fat cells, and the ability of exogenously added GM1 to enhance the toxin's lipolytic activity represents the specific creation of additional natural receptors on adipocytes. Alternative explanations are proposed which do not invoke GM1 as the native ***receptor*** for CT but which may account for the observed effects.

II LIPOLYTIC ACTION OF CHOLERA TOXIN ON FAT CELLS REEXAMINATION OF THE CONCEPT IMPLICATING GM-1 GANGLIOSIDE AS THE NATIVE MEMBRANE

RECEPTOR .

- AB. . . on isolated [rat] fat cells was examined. Analyses of the ganglioside content and composition of intact fat cells, their membranous ***ghosts*** and the total particulate fraction of these cells indicate that N-acetylneuraminylgalactosylglucosylceramide (GM3) represents the major ganglioside, with substantial amounts of. . . .times. 10⁹ molecules were associated with the cells. Essentially all exogenously added GM1 was found bound to the plasma membrane ***ghost*** fraction. Investigation of the biological responses of the cells confirmed their sensitivities to CT and epinephrine-stimulated lipolysis, as well as. . . . pronounced than, that of GM1. These findings do not support the earlier hypotheses that GM1 is CT's naturally occurring membrane ***receptor*** on native fat cells, and the ability of exogenously

added

GM1 to enhance the toxin's lipolytic activity represents the specific creation of additional natural receptors on adipocytes. Alternative explanations are proposed which do not invoke GM1 as the native

receptor for CT but which may account for the observed effects.

ORGN Classifier

Bacteria 05000

Super Taxa

Microorganisms

Taxa Notes

Bacteria , Eubacteria, Microorganisms

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates,. . .

L7 ANSWER 62 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

AN 1977:125713 BIOSIS <<LOGINID::20091202>>

DN PREV197763020577; BA63:20577

TI MUTATIONAL CHANGE OF MEMBRANE ARCHITECTURE MUTANTS OF ESCHERICHIA-COLI K-12 MISSING MAJOR PROTEINS OF THE OUTER CELL ENVELOPE MEMBRANE.

AU SCHWEIZER M; SCHWARZ H; SONNTAG I; HENNING U

SO Biochimica et Biophysica Acta, (1976) Vol. 448, No. 3, pp. 474-491.

CODEN: BBACAQ. ISSN: 0006-3002.

DT Article

FS BA

LA Unavailable

AB Mutants of E. coli were analyzed which lack 2 of the major proteins of the outer cell envelope membrane. The 2 proteins I and II*, normally are present at high concentrations (.apprx. 105 copies/cell). In such mutants, as compared with wild type, the phospholipid-to-protein ratio in the outer membrane increased by a factor of 2.3, causing a considerable difference in density between wild type and mutant membranes. The concentrations of 2 other major components of the outer membrane, lipopolysaccharide and Braun's lipoprotein, did not change. The protein-deficient mutants do not exhibit gross functional defects in vitro. An increased sensitivity to EDTA and a slightly increased sensitivity to dodecyl sulfate (but not to deoxycholate or Triton X-100) was observed, loss of periplasmic enzymes was not found, and other differences to wild type are marginal. The mutants grow with normal morphology. It is not possible to prepare ***ghosts*** (particles of size and shape of the cell without murein, surrounded by a derivative of the outer membrane, and possessing the major proteins of this membrane) from them. The proteins in question are apparently required for the shape maintenance phenomenon in ***ghosts***, and the mutants indicate that these proteins are not involved in the expression of the genetic information specifying cellular shape. Freeze-fracturing showed that in mutant cells, in sharp contrast to wild type, the predominant fracture plane is within the outer membrane. The concentration of the densely packed particles at the outer and concave leaflet of this fracture plane is greatly reduced. It was not possible to establish that 1 or the other protein is part of these particles since these ultrastructural differences were not apparent in mutants missing only 1 of the proteins. The loss of 2 major proteins and the concomitant increase of phospholipid concentration apparently changed the architecture of the outer membrane from a highly oriented structure with a large fraction of protein-protein interaction, to 1 predominantly exhibiting planar lipid bilayer characteristics. E. coli thus assemble different outer membranes,

indicating that outer membrane formation does not constitute a highly ordered or strictly sequential assembly-line process.

AB. . . and other differences to wild type are marginal. The mutants grow with normal morphology. It is not possible to prepare ***ghosts*** (particles of size and shape of the cell without murein, surrounded by a derivative of the outer membrane, and possessing. . . major proteins of this membrane) from them. The proteins in question are apparently required for the shape maintenance phenomenon in ***ghosts*** , and the mutants indicate that these proteins are not involved in the expression of the genetic information specifying cellular shape.. . .

IT Miscellaneous Descriptors
 PHOSPHO LIPID LIPO POLY ***SACCHARIDE*** MUREIN PERIPLASMIC ENZYME
 MORPHOLOGY GENE EXPRESSION CONCAVE LEAFLET STRUCTURE

ORGN Classifier
 Bacteria 05000
 Super Taxa
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 63 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
 STN DUPLICATE 16

AN 1976:216651 BIOSIS <<LOGINID::20091202>>
 DN PREV197662046651; BA62:46651

TI MULTIPLE STEPS DURING THE INTERACTION BETWEEN COLI PHAGE LAMBDA AND ITS
 RECEPTOR PROTEIN IN-VITRO.

AU ROA M; SCANDELLA D
 SO Virology, (1976) Vol. 72, No. 1, pp. 182-194.
 CODEN: VIRLAX. ISSN: 0042-6822.

DT Article
 FS BA
 LA Unavailable

AB Phage .lambda. and its purified ***receptor*** protein [Escherichia coli] interact in vitro even when the phage is not inactivated; in the absence of detergent the ***receptor*** is relatively insoluble and it leads to the formation of phage aggregates. Under conditions where the phage is inactivated by the ***receptor*** , only a small fraction (about 30%) of its DNA becomes sensitive to nucleases. Ejection of the DNA apparently is almost complete upon sucrose gradient centrifugation since clear separation of ***ghosts*** and DNA can be obtained. It is possible to recover from the gradients some inactive phage particles which have not yet ejected their nucleic acid. The different steps occurring in vitro, i.e., reversible interaction, phage inactivation and DNA ejection, are correlated with the 1st steps of phage infection in vivo.

TI MULTIPLE STEPS DURING THE INTERACTION BETWEEN COLI PHAGE LAMBDA AND ITS
 RECEPTOR PROTEIN IN-VITRO.

AB Phage .lambda. and its purified ***receptor*** protein [Escherichia coli] interact in vitro even when the phage is not inactivated; in the absence of detergent the ***receptor*** is relatively insoluble and it leads to the formation of phage aggregates. Under conditions where the phage is inactivated by the ***receptor*** , only a small fraction (about 30%) of its DNA becomes sensitive to nucleases. Ejection of the DNA apparently is almost complete upon sucrose gradient centrifugation since clear separation of ***ghosts*** and DNA can be obtained. It is possible to recover from the gradients some inactive phage particles which have not. . . .

ORGN Classifier

Viruses 03000
 Super Taxa
 Microorganisms
 Taxa Notes
 Microorganisms, Viruses
 ORGN Classifier
 Bacteria 05000
 Super Taxa
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 64 OF 68 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on
 STN
 AN 1978:66865 BIOSIS <<LOGINID::20091202>>
 DN PREV197815010365; BR15:10365
 TI ULTRASTRUCTURAL AND BIOCHEMICAL EXAMINATIONS OF CELL WALL ***GHOSTS***
 OF ROUGH AND SMOOTH STRAINS OF ESCHERICHIA-COLI.
 AU LAM J; IRVIN R T; COSTERTON J W
 SO Canadian Federation of Biological Societies Proceedings, (1976) Vol. 19,
 pp. 12.
 ISSN: 0068-869X.
 DT Article
 FS BR
 LA Unavailable
 TI ULTRASTRUCTURAL AND BIOCHEMICAL EXAMINATIONS OF CELL WALL ***GHOSTS***
 OF ROUGH AND SMOOTH STRAINS OF ESCHERICHIA-COLI.
 IT Miscellaneous Descriptors
 ABSTRACT FREEZE ETCHING LIPO POLY ***SACCHARIDE*** OUTER MEMBRANE
 ORGN Classifier
 Bacteria 05000
 Super Taxa
 Microorganisms
 Taxa Notes
 Bacteria , Eubacteria, Microorganisms

L7 ANSWER 65 OF 68 MEDLINE on STN
 AN 1975046845 MEDLINE <<LOGINID::20091202>>
 DN PubMed ID: 4139715
 TI Use of the ***avidin*** - ***biotin*** complex for specific staining
 of biological membranes in electron microscopy.
 AU Heitzmann H; Richards F M
 SO Proceedings of the National Academy of Sciences of the United States of
 America, (1974 Sep) Vol. 71, No. 9, pp. 3537-41.
 Journal code: 7505876. ISSN: 0027-8424.
 Report No.: NLM-PMC433809.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 197501
 ED Entered STN: 10 Mar 1990
 Last Updated on STN: 6 Feb 1995
 Entered Medline: 25 Jan 1975
 AB To expand the electron microscopist's options in localization and
 visualization, a new and general staining technique has been tested. The
 avidin - ***biotin*** complex serves as a coupling between the

electron-dense marker, ferritin, and points of interest in biological samples. When specific cellular components are tagged with ***biotin***, those components may be visualized with ferritin-linked ***avidin***. Because of the remarkably strong affinity of ***avidin*** and ***biotin*** (characterized by an association constant of $10(15) \text{ M}^{-1}$), the staining is rapid and stable. The preparation of ferritin-***avidin*** conjugate is described, and examples are presented of the application of this complex to ***biotin***-tagged membranes. The ***ghosts*** of *Acholeplasma laidlawii* have been treated with biotinyl-N-hydroxysuccinimide ester to label protein amino groups. Erythrocyte membrane oligosaccharides have been oxidized by periodate or by galactose oxidase, and the resulting aldehydes labeled with ***biotin*** hydrazide. The ***avidin*** - ***biotin*** complex in electron microscopy seems especially appropriate for sequential staining procedures, as well as for visualization of reaction sites of ***biotin***-labeled, low-molecular-weight reagents.

TI Use of the ***avidin*** - ***biotin*** complex for specific staining of biological membranes in electron microscopy.

AB . . . To expand the electron microscopist's options in localization and visualization, a new and general staining technique has been tested. The ***avidin*** - ***biotin*** complex serves as a coupling between the electron-dense marker, ferritin, and points of interest in biological samples. When specific cellular components are tagged with ***biotin***, those components may be visualized with ferritin-linked ***avidin***. Because of the remarkably strong affinity of ***avidin*** and ***biotin*** (characterized by an association constant of $10(15) \text{ M}^{-1}$), the staining is rapid and stable. The preparation of ferritin-***avidin*** conjugate is described, and examples are presented of the application of this complex to ***biotin***-tagged membranes. The ***ghosts*** of *Acholeplasma laidlawii* have been treated with biotinyl-N-hydroxysuccinimide ester to label protein amino groups. Erythrocyte membrane oligosaccharides have been oxidized by periodate or by galactose oxidase, and the resulting aldehydes labeled with ***biotin*** hydrazide. The ***avidin*** - ***biotin*** complex in electron microscopy seems especially appropriate for sequential staining procedures, as well as for visualization of reaction sites of ***biotin***-labeled, low-molecular-weight reagents.

CT *Acholeplasma laidlawii*: CH, chemistry
 ****Avidin***
 *** Bacterial Proteins: AN, analysis***
 ****Biotin***
 Carbohydrates: AN, analysis
 Carbon Radioisotopes
 Erythrocytes: CH, chemistry
 Ferritins
 Immunodiffusion
 Ligands
 *Membranes
 Membranes: CH, chemistry
 *Microscopy, Electron: MT, methods
 *Ovalbumin

. . .
 RN ***1405-69-2 (Avidin)*** ; ***58-85-5 (Biotin)*** ; 9006-59-1 (Ovalbumin); 9007-73-2 (Ferritins)
 CN 0 (***Bacterial*** Proteins); 0 (Carbohydrates); 0 (Carbon

Radioisotopes); 0 (Ligands); 0 (Proteins)

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STN
AN 1974:132516 BIOSIS <<LOGINID::20091202>>
DN PREV197457032216; BA57:32216
TI CELL ENVELOPE AND SHAPE OF ESCHERICHIA-COLI STRAIN K-12 THE ***GHOST***
MEMBRANE.
AU HENNING U; HOEHN B; SONNTAG I
SO European Journal of Biochemistry, (1973) Vol. 39, No. 1, pp. 27-36.
CODEN: EJBCAI. ISSN: 0014-2956.
DT Article
FS BA
LA Unavailable
TI CELL ENVELOPE AND SHAPE OF ESCHERICHIA-COLI STRAIN K-12 THE ***GHOST***
MEMBRANE.
IT Miscellaneous Descriptors
PHOSPHO LIPID LIPO POLY ***SACCHARIDE*** PROTEINS MUREIN
ORGN Classifier
Microorganisms 01000
Super Taxa
Microorganisms
Taxa Notes
Microorganisms
ORGN Classifier
Bacteria 05000
Super Taxa
Microorganisms
Taxa Notes
Bacteria , Eubacteria, Microorganisms

L7 ANSWER 67 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1970:98551 CAPLUS <<LOGINID::20091202>>
DN 72:98551
OREF 72:17865a,17868a
TI Specific inhibition of endotoxin coating of red cells by a human
erythrocyte membrane component
AU Springer, Georg F.; Huprikar, Shankar V.; Neter, Erwin
CS Dep. of Immunochem. Res., Evanston Hosp., Evanston, IL, USA
SO Infection and Immunity (1970), 1(1), 98-108
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB A fraction from human erythrocyte ***ghosts*** was isolated which
prevents the attachment of unheated as well as heated lipopolysaccharides
of gram-neg. ***bacteria*** to red cells. This material has no
significant inhibitory effect either toward the Vi antigen of gram-neg.
bacteria or towards the group and common antigens of the gram-
pos.
bacteria investigated. It interacts with lipopolysaccharides and
not with erythrocytes, it forms complexes with and blocks those groupings
of lipopolysaccharides which attach to red cells. The effect of the
receptor is phys. and not enzymic. The interaction of the
receptor with the lipopolysaccharides is reversible, and the
receptor removes lipopolysaccharides fixed to red cells. An
equil. of lipopolysaccharide distribution between cells and

receptor is established when ***receptor*** -
 lipopolysaccharide
 complexes are incubated with red cells. The ***receptor*** is labile
 toward heat and toward deviation of the H+ concn. from neutrality;
 aldehydes destroy its inhibitory activity.

AB A fraction from human erythrocyte ***ghosts*** was isolated which
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 pos.

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 receptor removes lipopolysaccharides fixed to red cells. An
 equil. of lipopolysaccharide distribution between cells and
 receptor is established when ***receptor*** -
 lipopolysaccharide
 complexes are incubated with red cells. The ***receptor*** is labile
 toward heat and toward deviation of the H+ concn. from neutrality;
 aldehydes destroy its inhibitory activity.

IT Toxins
 RL: PROC (Process)
 (erythrocyte binding of, ***receptor*** in)

IT ***Bacteria***
 (lipopolysaccharides of gram-neg., erythrocyte ***receptor*** for)

IT Lipopolysaccharides
 RL: PROC (Process)
 (of ***bacteria*** , erythrocyte binding of)

IT Erythrocytes
 (toxin binding by, ***receptor*** in)

L7 ANSWER 68 OF 68 CAPLUS COPYRIGHT 2009 ACS on STN
 AN 1970:108292 CAPLUS <<LOGINID::20091202>>
 DN 72:108292
 OREF 72:19565a,19568a

TI ***Receptor*** specificity during the interaction of Escherichia coli
 bacteria with "shades" of T4 phage

AU Guseinov, R. D.
 CS Inst. Obshch. Genet., Moscow, USSR
 SO Doklady - Akademiya Nauk Azerbaidzhanskoi SSR (1969), 25(7), 72-6
 CODEN: DAZRA7; ISSN: 0002-3078

DT Journal
 LA Russian

AB The shades of T4 phages are able to kill only sensitive cells. Their
 action spectrum is the same as that of intact phage particles. E. coli
 strain B/4 resistant to T4 phage was changed to a sensitive one after
 10-min treatment with EDTA at 37.degree.. Various hypotheses of reaction
 mech. are discussed.

TI ***Receptor*** specificity during the interaction of Escherichia coli
 bacteria with "shades" of T4 phage

IT Viruses, ***bacterial***
 (T 4, Escherichia coli interaction with ***ghosts*** of,
 receptor specificity in)

IT Escherichia coli

```
(  ***bacteriophage***      ***ghost***  interaction with,  
  ***receptor***    specificity in)
```